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The Second South African Medico-Legal Congress

The First South African Medico-Legal Congress was held in Johannesburg in 1958 under the distinguished patronage of the then Chief Justice, the Honourable Mr. H. A. Fagan, with Prof. S. F. Oosthuizen (President of the South African Medical and Dental Council) as President. The Congress was an unqualified success from the standpoint of legal as well as medical practitioners.

The initiative of the South African Medico-Legal Society in sponsoring and convening this First Forensic Conference created a new pattern for forensic meetings in South Africa and has resulted in a decision to hold a Second South African Medico-Legal Congress in Durban from 4–7 July 1960.

It is anticipated that distinguished forensic pathologists from overseas will participate in the meeting, which will also be attended by Sir Herbert Edmund Davies, a leading British judge who recently presided over the Podola trial, when amnesia as a defence was so thoroughly canvassed.

The detailed programme of the Durban meeting is not yet available. It is known, however, that it will include a series of plenary sessions which will be of great interest to lawyers as well as doctors. A feature of the occasion will be the inclusion of daily medico-legal autopsy demonstrations which should be of great value, particularly to district surgeons in practice in rural areas throughout South Africa.

The Second Congress is being held in association with the jubilee celebrations of the University of Natal. The Honorary Organizing Secretary is Prof. I. Gordon, Dean of the Faculty of Medicine of the University of Natal, Durban. Those interested in receiving further particulars should write to him without delay, especially in connexion with the reservation of accommodation.

The Second International Meeting on Forensic Pathology

The Second Meeting will be held in New York, N.Y., U.S.A., from 18 to 21 September 1960. The Conference will be associated with the dedication of the new building for the office of the Chief Medical Examiner of the City of New York.

Further information may be obtained from any of the following: Dr. Milton Helpern, 55 East End Avenue, New York 28, New York, U.S.A., *or* Dr. Charles Larsen, Tacoma General Hospital, Tacoma 5, Washington, U.S.A., *or* Dr. Francis E. Camps, 37 Welbeck Street, London, W.1, England.

Evaluation of Disability*

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In accidental injury cases, whether the claims are made in the civil courts or under the Workmen's Compensation Act, the question of *liability* is usually decided by the judge or the Workmen's Compensation Commissioner's staff without the help of outside experts. On the question of damages however there is a substantial contribution of information and opinion to be contributed by doctors and other experts.

Apart from expressing an opinion on temporary total disablement with total loss of earnings, and temporary partial disablement with partial loss of earnings, the doctors have to describe as far as possible the pain and suffering occasioned by the consequences of the accident.

Careful assessment of past, present and future pain and suffering requires clinical familiarity with the conditions found. Similarly an expression of opinion on the diagnosis, its probable duration, its possible cure and its effect on expectation of life fall within the doctor's scope. But the most important aspect of the doctor's report covers an assessment of permanent disablement and its effect on future earning capacity and possible loss of amenities. When there are profound differences of opinion amongst the medical experts, the judge may well be misled or confused and yet has to make a decision in a sphere in which he is not necessarily well-informed.

Fortunately by now the legal profession appreciates that there are many problems in medicine in which no clearcut opinion is possible.

Doctors of varying degrees of skill, experience and impartiality have to express opinions on problems under cross-examination. It is small wonder that opinions are sometimes expressed a little more strongly than the facts or the state of medical knowledge warrant. Reservations are sometimes omitted because the opportunity of making them is missed.

It could not be denied that partiality may be subtle and elusive and not exposed in court.

In the evaluation of disability, one of the main functions of the examining doctor is to detect errors and false statements in the factual findings and opinions expressed. In work of this sort, most mistakes arise from inadequate examination, misinterpretation of clinical findings, errors in X-ray technique or interpretation and misapplying references to technical literature.

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*An address delivered at a meeting of the South African Medico-Legal Society on 5 August 1959 at 8 p.m. in the Harveian Theatre, Medical School, University of Witwatersrand, Johannesburg.

The charitable interpretation of such errors is that of ignorance. Probably, however, it is more often due to the inherent lack of thoroughness that may characterise a man's work in any field.

Lawyers frequently warn their clients to refuse to answer questions relating to the circumstances of the accident. One must admit that medical reports sometimes contain a great deal of information on the history of the accident, which might affect the merits of the claim. But the doctor should be careful to limit his questions in such a way that it is clear that he is investigating the applied mechanics of the accident. He is entitled to know a great deal about the exact nature of the violence applied to the injured person at the moment of impact and afterwards. Similarly he is entitled to discover how much the injured person remembers of the events preceding the accident. After all, the existence of amnesia for a period before the collision is of neurological significance.

He has to express an opinion whether the nature of the injuries is consistent with the severity of violence applied to the victim.

In the same way, a repetition of the medical history of the case is necessary in the report. Although it is hearsay information, it does represent what the victim remembers of his pain, suffering and treatment. In this respect it should be noted that most of the clinical information available to the psychiatrist is second-hand and not derived from direct observation.

The doctor as a clinician has to express his opinion not only on what he finds on physical examination, but also on what the patient supplies in the way of complaints, symptoms and imaginary complaints. If the doctor were not free to question the patient adequately he would, for practical purposes, be in the position of a veterinary surgeon examining a horse.

If the expert is to be truly helpful he should be permitted to consider all factors which have contributed to the past or present disability of the injured person.

In my opinion psychiatric examiners should be allowed considerable latitude in this respect.

Workmen's Compensation and invalidity insurance and invalidity grants from the State require a working definition of the incapacities for which benefits are payable.

In many countries information is sought concerning definitions of incapacity, methods of evaluation and incapacity schedules. Schedules are often inappropriate because of the diversity of physical types, the different possibilities of rehabilitation and the unequal duration of expected life, especially working life.

There are three concepts of incapacity, viz. physical invalidity, occupational incapacity and general incapacity for work.

The severity of physical damage of itself cannot afford an adequate basis for the evaluation of incapacity, and under most Workmen's Compensation Acts forms but one of the factors taken into account in a valuation. The question is whether the loss of earning capacity should be assessed with reference to the pre-accident occupation or to possible earning capacity on the open labour market, perhaps after rehabilitation.

Further, it would appear to be unfair to estimate incapacity for work in total disregard of the employment situation.

In most countries this complicated problem has been reduced to an arbitrary Workmen's Compensation Act Schedule or Military Pensions Schedule which is applied largely by push-button methods.

In some countries these schedules are hard and fast with limited possibilities of variation and review. In other countries the schedules are only a guide indicating minimum percentage estimates for stated anatomical loss and considerable discretion is allowed for increases in special circumstances.

Before a disability can be evaluated, obviously a norm should be determined.

The International Labour Office at Geneva made a summary of dozens of different schedules of disabilities from many countries and demonstrated gross differences of opinion and custom in assessments.

In this country the Military Pensions Schedule differs considerably from the Workmen's Compensation Act Schedule which, in turn, differs markedly from Schedules in other countries.

Even in the United States each of 48 States has its own schedule and an attempt is now being made at drafting a consolidating schedule.

All these Workmen's Compensation Act schedules compare the disabled person to that nebulous imaginary person—the 'workman as defined' in the Workmen's Compensation Act. This mythical person is supposed to work in the *open labour market*. As everyone knows, the market is not open and an electrician may not do the work of a carpenter or a plumber, each being specialists in respective divisions of the open labour market. But the assessment of the disability of an injured workman is supposed not to be related to his particular employment. Only in exceptional circumstances does the Commissioner exercise the special powers vested in him and raise the permanent disability assessment in relation to the man's particular employment.

This anomaly in our Workmen's Compensation Act is manifestly unfair to the workman, e.g. a disabling amputation would severely disable a rigger, linesman or train conductor, but would be much less disability to a works manager or a clerical worker.

Bearing in mind the prolonged training of artisans, it appears to me that assessment should be specifically related to the man's trade or professional skills, or to the transferability of his skills to another occupation.

Schedules of Disablement

Persons with mathematical minds have attempted to break up the whole into composite parts and to allocate arbitrary percentage values to anatomical appendages.

McBride, e.g. has attempted a mathematical analysis of the balanced factors that create the ability to work.

I. *Functional ability is divided into*:

1. Quickness of action.
2. Co-ordination.
3. Strength.
4. Security.
5. Endurance.
6. Safety as workman.
7. Prestige of physique for employment.

If the percentage loss under each heading is averaged it will yield the percentage disability for the functional deficiency component.

II. *The physical disorders component is divided into*:

1. Anatomical mass damage.
2. Clinical manifestations.
3. Restrictions of physical requirements.
4. Restriction of working conditions.
5. Reactionary factors.

1. *Anatomical Mass Damage.* This is the basic source of disability. The extent is determined by thorough diagnostic examination and requires knowledge of anatomy, physiology, pathology and body mechanics. For example, a fractured shaft of femur may change the length, alignment, contour and mechanics of the bone as well as alter the mechanics of the attached muscles and ligaments. The related joint areas may be altered, kinetic forces lessened, long axis support lessened and the line of equilibrium upset. Such factors as malunion, non-union, shortening and angulation are commonly present.

2. *Clinical Manifestations.* These are the result of anatomical change. The intensity of the symptoms is not necessarily indicative of the magnitude of the anatomical damage. A small lesion may be grossly disabling. Permanent manifestations should be clearly differentiated from temporary symptoms and findings.

Such factors as pain, muscle spasm, loss of motion, paresis, fibrotic contracture, muscle atrophy, disturbance of articulation and of sensation are evaluated.

In this way an estimate of percentage loss is made under this item.

3. *Restriction of Physical Requirements.* The greater the opportunity for employment, the less severe is the physical incapacity for work. A stiff joint may be more disabling than an amputation. The extent to which such factors restrict restoration to employment is assessed as an item.

4. *Restriction of Working Conditions.* Exposure to temperatures, dust, chemicals or slippery floors may limit the man's potential field.

5. *Intangible Reactionary Interferences.* The reactionary influences involving mental, social and economic impediments are difficult to determine. Most disabled persons are anxious to rehabilitate themselves, but others require assistance to do so. This assistance may not be forthcoming. A man may be declared fit for light work but the employer may say there is no light work available.

This impasse is an everyday one in industry. The man can hardly retain his self-respect when he returns to work with a partially disabled back and is placed on a job requiring heavy lifting. The age factor may be a strong factor in reducing a man's chances of re-employment.

Taking these five factors, each on a percentage basis, the average is obtained under the item of physical disorder. McBride has evolved a formula on which the functional disability carries three times the significance of the physical disorder component.

I am not advocating this method, but I have quoted it in an abbreviated form to indicate how complicated such an attempted mathematical breakdown can become.

When doctors accustomed to making assessments examine a disabled man these factors are undoubtedly applied in obtaining an overall clinical impression of the man's disability.

In published schedules for different countries, the anatomical and functional deficiencies related to these bluntly stated figures are implied. For example, if a below-knee amputation is rated at 35% permanent disability, it already means that apart from anatomical loss, the loss of quickness of action, co-ordination, strength, security, endurance, prestige of physique have similarly been considered.

Therefore these schedules are by no means as lifeless and unrealistic as is often argued.

Our own Workmen's Compensation Act schedule could, of course, be made much more comprehensive. This has not been done because up to the present no Committee has yet succeeded in agreeing sufficiently for an acceptable schedule to be submitted for consideration by the Commissioner.

Multiplicity

The effects of certain multiple injuries are greater than the mathematical sum of the separate factors. For example, a man with a stiff hip may be 20% disabled. With a stiff knee on the same side which alone would merit an assessment of 20%, he is however more disabled than 40%. He would probably be at least another 10% disabled, i.e. 50%.

Industrial Disability Evaluation

Diseases and injuries arising from industrial activity have always presented a major problem and the laws under which they have been managed have been radically changed and progressively improved for many years. At the present time in most civilized countries workmen are receiving very much better industrial surgical care than ever before, but in spite of that, new difficulties have developed and the methods of management of industrially injured persons have changed gradually; in this country I believe further change may be anticipated. At the present time, the injured workman receives periodical payments calculated on a formula during his period of total temporary disablement. At the same time, he draws benefits from various sources, such as the Unemployment Fund, and in many cases is as well off financially while he is off work as he is normally at work. In the case of workmen carrying private insurance, some of them are better off while they are not working.

Whereas initially the attendance of injured workmen was more or less arbitrarily restricted to so-called factory surgeons of the employers' choice, it has now been extended to include almost the entire medical profession, and general practitioners as well as specialists of all kinds commonly participate in this ever-increasing medical service. The patient exercises his own judgment in the selection of his doctor who generally advises him if and when specialized service is required and who shall provide it.

This places the responsibility for the entire system of medical care of the industrially injured workman upon the doctor who is first consulted in the case, and much attention is now being directed towards an evaluation of his effectiveness in this role. In fact it is quite well known that the average practitioner, having recently survived an intensive course of seven years of concentrated medical training, has not had time to study adequately the special problems involved in dealing with injured workmen. Fortunately the medical curriculum is so designed as to produce a very humble trainee at the end of the seven years, a man who recognizes his limitations and who should not hesitate to seek suitable assistance when required. This assurance, for what it is worth, is of importance to the insurance carrier, the industrialist who pays the premium on the insurance policy and the workman who ultimately has to benefit from treatment and rehabilitation.

If we may learn from the haphazard development of Workmen's Compensation services in more advanced countries, it would be of assistance to refer briefly to an interesting study of the subject by Henry Kessler, who investigated the problem of low back pain in industry under the auspices of the Commerce and Industry Association of New York Incorporated in 1955. Some fantastic facts were established. In the state of New York in 1952, of the 700,000 cases adjudicated, 23,000 were cases of low back pain. Those cases suffering from fractures of the spine averaged a period off work of 232

weeks. This was in sharp contrast to common medical experience in New York where, in non-industrial cases, the period for comparable cases was 24 weeks. Thus under the Compensation Act in the state of New York the man with the fractured back was disabled on an average at least ten times longer than he would have been had his injury occurred under non-compensable circumstances. It is evident, therefore, that the Compensation Act as it functions in that State, is more of an economic detriment than a boon to the injured workman. The money incentive behind workmen's compensation and the failure of all those who participate in its operation to visualize its true objective to reduce the period of incapacity and to quickly and fully restore the workman to his job are the blights that produce all these problems.

In the same study, it was attempted to discover which factors were significant in prolonged disability and which one of these significant factors was dominant in prolonging disability.

The effects of numerous specific factors were observed. The socio-economic factors of age, sex, location of employment, pre-employment condition and wage rates were noted, also the administrative features, such as the duration of the administrative process, the number of hearings, the frequency of hearings, the medical costs, compensation costs and the current status of the case. Finally the medical factors including the type of the first attending physician, the presence of a pre-existing back condition, the number of recurrences, the number of physicians, the elapsed time between the first referral to an orthopaedic surgeon, the nature of the medical treatment such as bed rest, traction, manipulation, physiotherapy and surgery.

It was surprising to note that in none of these groups of factors could a single one be found which was sufficiently significant by itself to be a determinant in prolonging disability. However, clinical evaluation showed that concealed in these specific factors was a hidden one, i.e. the total effect of all these factors impinging on the personality and constitution of the individual worker. It was this very multiplicity and complexity of factors and the attention, confusion and bewilderment which could be summed up in the term 'compensation' system which must be indicted as the causative factor responsible for the production of much of the neurotic behaviour on which the prolonged disability was based. In a detailed analysis of 160 cases of backache, it was proved that in 20% of cases the findings were consistent with degeneration of a lumbar intervertebral disc, while in the remaining 80% it was characterized by an anxiety state predicated on the presence of multiple complaints not related to the back.

Psychiatric findings of intrinsic factors in the patient's personality and extrinsic factors related to litigation, all of which resulted in failure to achieve rehabilitation up to the time of a final disposition of the claim and sometimes even afterwards. It was noteworthy that all the standard methods

of treating low back pain fell short of their objective in these problem cases. Even surgery undertaken late was generally futile. Thus it would appear that the intractable injury is not one confined to the anatomy of the low back alone, but related to the total personality and all the intrinsic and extrinsic forces which influence it under the compensation system. This explains why the termination of the compensation aspect of the problem represented the only effective means of solving some of the most intractable cases.

It will be apparent from the foregoing that the basic failure has been in the inadequate evaluation of disability and adjusting the compensation machine to such scientific evaluation. In other words, if it is possible to find precisely what the man's disability really is, it becomes possible to deal with him, and failure at the early evaluation stage results in a breakdown of the whole mechanism. In recent years Workmen's Compensation laws have increasingly shifted the emphasis from compensation to prevention, treatment and particularly rehabilitation. Although compensation is important to the individual workman, especially during the period of his total inability to work, prevention, adequate treatment and maximum rehabilitation are even more important, not only to him but also to the community. It serves little purpose to substitute monetary benefits for functional and vocational usefulness. The money offered rarely even closely approximates what the patient would have been able to earn were he not incapacitated and is no substitute for the self-sufficient, useful worker who continues to contribute his share to the total economy. Therefore the primary goal for the injured worker should be rehabilitation rather than compensation. The problem that confronts the doctor in his management and appraisal of a person with a low back injury involves many factors which require consideration in establishing an opinion which will fairly assess the merits of the case.

Many outstanding experts have tackled the subject and many different methods of computation have been evolved. The subject is a difficult one and the more it is studied, the more elusive it seems to become. After long years of experience with large numbers of cases some form of rationalization develops out of apparent chaos. I sometimes think that doctors applying simple rules of thumb do better than those who have philosophic doubts about a multiplicity of aspects of the difficult cases.

In Ontario a method of dealing with back cases has been evolved. It is as follows:

1. The rating of permanent disability in postoperative disc claims is to be based on two factors, viz. actual physical disability as determined by physical examination; and the occupational handicap as determined by the type of worker the injured person is, what he did before the accident and the extent of his restoration to industry.
2. When he is a workman who is able, without loss of earnings, to perform the duties of his former or similar occupation, even though he has some discomfort in his back at times, no permanent disability award is to be made, but his case remains open for future reconsideration in the event of a change in his industrial status attributable to the original low back injury.

3. Where an injured workman has to accept lighter employment because it is either detrimental or impossible to perform the heavy duties involved in his former occupation a permanent award is to be considered even though, on physical examination, the evidence of actual physical disability is minimal.
4. Where there is definite evidence of physical disability on physical examination, the factor of occupational handicap is to be considered in conjunction with it.

This method is similarly open to serious criticism because it only solves the disability problem in one type of case where there are no significant post-traumatic physical defects and the workman returns to his former vocational status even though still suffering from pain.

It is significant that in the United States of America there is a separate Workmen's Compensation Act Schedule of Disabilities in each State, and the methods of assessment and settlement vary from State to State. Repeated attempts at unifying and standardizing legislation on this issue have so far failed, although a further determined attempt is being made at the moment.

Stage of Evaluation of Disability

At the time of examination the disabled person may not have reached the stage of a stable disablement, e.g. in Workmen's Compensation Act work it was felt that speedy settlement of a claim meant a satisfied worker. Delay in assessment piles up the cost of medical care, Unemployment Act benefits, periodical payments of part income and, in practice, was found to involve higher assessments.

In this country industrial assessments are at present relatively free of the troubles found in New York by Kessler. Our Workmen's Compensation Commissioner (notwithstanding shortage of skilled clerical and medical staff) manages to give the injured workman a square deal with less loss of time and money and temper than is possible in New York. However, as legal and medical colleagues we will have to guard against a change in this respect.

For example, if the autonomy vested in the Workmen's Compensation Commissioner were surrendered to a Government Department such as the Department of Labour, I personally would predict a serious deterioration in the quality of service rendered to the injured workman and the employer.

To put it bluntly, I believe that political pressure does not at present play a significantly harmful part in Workmen's Compensation Assessments and I hope it will not do so in the future.

To revert to my original point, that of stage of assessment:

Theoretically, while active changes are occurring in the workman's condition, his final state cannot be judged. In practice, however, this is done daily both in Workmen's Compensation and civil claims.

In fact, often an opinion is expected about the man's probable state after further operation and rehabilitation. In this way many assessments

are predictions of probabilities and not remotely related to the ultimate truth, e.g.:

I went fishing with a man at St. Lucia one year. He had had 7 fractured vertebrae and had been paid out 100% disability by the Mines some years earlier. He jumped about on the rocks like a cat on hot bricks, spent the day spinning for barracuda and kingfish all day, and never complained of a backache.

I met an old patient in Eloff Street one day. He was so proud of his physical fitness, that I took a cine-film of him doing cartwheels and hop-skip-and-jumps on my lawn. A few years before he had been assessed at 60% disabled for two fractured thighbones and two fractured ankles. Yet he was drawing a pension for life.

In our Workmen's Compensation Act disablement is defined as 'disablement for employment or permanent injury or serious disfigurement.' Loss of earnings is therefore not necessarily an essential element in entitlement to compensation.

Apparently the treating doctor should base his estimate of permanent disablement on the medical aspects of disablement, without the skilled guidance of an investigating officer. It is not within the competence of the clinician to assess the economic consequences of a particular case, i.e. under the Workmen's Compensation Act.

In determining disablement, loss of function or inability 'to do' should be taken as a measure of disablement for the open labour market which is the background against which the disability has to be considered.

In assessing inability 'to do' the different components of function, e.g. movement, speed, power, co-ordination, stability, skill, sensitivity, prehensile ability, etc. should be consciously evaluated in an attempt at objectivity.

Where a condition has specifically serious consequences, the Workmen's Compensation Commissioner may award a special percentage of permanent disablement and it is permissible for the doctor to draw his attention to this position—rarely done in practice.

In cases that are likely to alter materially and where the disability is in excess of 25%, a provisional pension can be awarded with review at a later stage. This obviates payment of a lump sum which may later be irrecoverable if permanent disablement is reduced materially.

Causal Relationship Between Injury and Disability

In the early stages of dealing with a severely injured person, this point is often neglected. It is always necessary to determine the causal relationship between a recognized injury and the compensable disability that arises out of it. The injured workman is invariably convinced that all his disabilities arise from the accident. It is a normal human tendency to attempt to explain physical disorders on the basis of some simple incident. The expectation of compensation and social security while under treatment may contribute to establishing in the worker's mind the causal relationship between the injury and the disability.

In many instances an employee cannot recognize the true nature of his disease and in good faith blames an incident at work. By the time he reaches the physician, he is able to explain his complaints with more or less precise details of the story of the accident and, if there is a delay of a few weeks, the story becomes so ingrained and crystallized in shape, that he believes it himself as a statement of the whole truth.

The man who bumps his shin in the workshop and is subsequently found to have a cancer of the bone invariably ascribes the cancer to the bump. As far as I know, there is no relationship between injury and an osteogenic sarcoma of the tibia, a well-known type of bone cancer. Every week thousands of rugby players get severe blows on their shin bones and if trauma were a causative factor in bone cancer, a substantial proportion of them should be developing the disease, but this is not so. Nevertheless, when a workman bumps his shin against an anvil in a workshop and he gets a bone cancer, I would say that the probabilities are that the accident fund is likely to accept such a relationship between the injury at work and the bone cancer, that the man will be given the benefit of such infinitesimal doubt as exists and will be paid out for loss of limb and ultimately of life.

This may be a very sympathetic attitude by an official in the head office of the accident fund, but it is very problematical whether any justification for such action can be found on scientific grounds. The doctor who may be asked to express an opinion would probably not be competent by training to express an opinion on such a matter and yet may be sufficiently casual in completing his certificate to give the impression that he feels there is a relationship between the injury and the cancer. In such circumstances serious injustice is done to the contributors to the fund—a fund which was not specifically designed as a charitable organization.

The study of case histories will show that many doctors are willing to accept the injured workman's view-point with regard to the causal relationship between the injury and the disease without critical examination of the facts. The physician firstly treats the pathological condition to the best of his ability. However, when treating a patient for injury under the Workmen's Compensation law, the physician also has a duty to the Accident Fund and indirectly to the community. A definite understanding of this relationship forms the necessary basis of an expert opinion in evaluating disability. The medico-legal aspects of injury or disease are foreign to many doctors. Many doctors attempt to steer clear of these considerations; others do do not realize that they are expected to be strictly impartial; yet other doctors believe that the task of determining compensability falls on the shoulders of the Insurance Carrier. This is a crucial point in the consideration of disabilities covered by the Workmen's Compensation Act. The most exact diagnosis of the consequences of injury can best be made very early after the accident and, very frequently, the clinical histories available given

immediately after the accident are short, inadequate and I would say given in a hurry or in circumstances not conducive to an objective evaluation of the problem in hand.

The relationship of trauma to a number of conditions requires further scientific investigation, but there is no real inducement for doctors to go off at a tangent to study these conditions; they are too busy dealing with sick people. Purely academic research is so poorly subsidized and, as far as I know, so inadequately supported by insurance carriers, that many of these problems have not been sufficiently elucidated. In every-day practice there are conditions such as 'frozen' shoulder, calcified tendons in the shoulder and the hip, rupture of the supraspinatus or biceps or quadriceps tendon, bursitis over the olecranon or the knee (so-called tennis elbow), damage to cartilages in the knee, varicose veins, cramps in the leg, pains in the feet, spurs on the heel bone, prolapsed disc, all sorts of backache, and sciatica, numbness and tingling in the fingers, diffuse discosis in the cervical spine. It is the responsibility of the doctor to analyse the causal relationship between such pathologies and the injury and frequently it boils down to the expression of opinion on general principles without very specific scientific work having been done on such a project. Insurance carriers pass on expenses to their consumers and therefore cannot have a very deep interest in such subjects. So long as somebody pays for it, it just increases the volume of business.

As far as I know the Workmen's Compensation Act does not cover the subsidy of medical research in fields such as this.

In determining the causal relationship between trauma and disease it must be decided which of a number of contributing factors is the important one. Failure to analyse the question along such lines is the chief pitfall in Workmen's Compensation procedure, as well as in insurance medicine in general.

The injured person, the doctor, the hospital and the insurance worker are all equally concerned. They all benefit from a prompt determination of the compensability of a given case. They are all embarrassed by a delayed decision.

The doctor requires diagnosis as exact and complete as possible to start effective treatment. The doctor should also be familiar with the working conditions under which the accident occurred.

The man's performance before the accident should also be known. He may have been less than normal before the accident.

Persons are employed indiscriminately or with inadequate pre-employment medical examination so that often enough the Insurance Carrier has bought a pig in a poke.

In the problem of backache, e.g. it is more than likely that many workmen would be classified as unacceptable as employees in heavy industry if their backs were examined beforehand.

The backs break down under conditions of stress. The insurance carrier is faced with an insoluble problem and ultimately pays for pre-existing disabilities and passes the buck by raising the cost of insurance.

This leads us to the consideration of aggravation.

Aggravation

Accidents occur in all stations of life and affect individuals who may be suffering from pre-existing disease or merely suffering from that degree of normal wear and tear and deterioration in the affected part which would be normal for that particular age group. For example, a man's knee joint is at its best when he is 18 years old and by the time he is 25 there are already early degenerative changes in the bearing surfaces of the knee joint. By the time he is 30 these changes are more gross; by the time he is 40 they are quite marked and by the time he is 60 he has worn away the white metal of the joint. In other words, the articular cartilage is gone and he is probably walking on bare bone.

This process of steady deterioration is much more marked than is generally appreciated, either by the medical profession or the lay public. If upon such deterioration a trauma is superimposed, according to the severity, more or less lasting effects may be produced. In fact, it is probable that once the degenerative process has reached a certain stage, even minor injury must produce permanent further deterioration which cannot be reversed by any process of treatment. Therefore the older the person is, the less is it possible to argue that the process produced by the trauma is reversible.

In the Workmen's Compensation Act there is no supposition that 'workmen as defined' should measure up to any particular standard of physical condition regarded as normal. It appears that employees should be taken as they are and if by reason of a physical defect an injury, which would not produce injury to a man in a normal state, results in severe disability or death, this result must be accepted as the legitimate consequence of an accident arising out of and in the course of employment, provided it can be shown that there has been material aggravation of the pre-existing condition. There must be some continuity of the thread of causation linking the disability or the death to the accident, which must have been severe enough to produce material aggravation.

However, it appears reasonable to assume that the employee must have been capable of a considerable degree of earning capacity prior to the accident. In other words, he must have been able to hold down his job. If such an employee is now injured in the course of his employment and the injury substantially permanently reduces his ability to do the same job, then the whole of the disability becomes compensable under the Workmen's Compensation Act.

It is obvious that in the assessment of such a disability a careful, previous history and record of the case are essential. Sometimes it is possible to compare an injured knee (with pre-existing arthritis which has been materially aggravated by trauma) with the other knee which has a similar pre-existing arthritis without injury; and the man's previous history may indicate that his knees were comparable in performance before the accident. Similarly good X-ray examination immediately after the accident, although it may reveal evidence of new injury, would also give an accurate picture of the condition of the knee at that time. When this is compared with subsequent X-rays, the deterioration in the injured joint may be compared and contrasted with the appearance of the other knee and of the injured knee at the time of the accident to determine the extent of the deterioration in the post-accident period.

The assessment of disability superimposed upon pre-existing disability pre-supposes an understanding of the normal biological progression of the pre-existing disease. Empirical estimates have to be made about how rapid the deterioration would have been had no injury occurred and compared to the aggravation or acceleration caused by the accident. It will be apparent that there is considerable scope for difference of opinion in a matter of this kind and that the examining surgeon's opinion will be very largely conditioned by his considerable experience of these biological processes.

In the aggravation of pre-existing disease, such as osteo-myelitis, osteo-arthritis, tuberculosis, etc. the time factor is important. For example, if a workman sustained a direct contusion to his hip joint and the next day is found to have active tuberculosis of the joint, then the accident cannot be blamed for it, although it may be possible to prove that the accident will accelerate the deterioration of the condition; but if there were an interval of, say, six weeks between the contusion and the manifested development of the tuberculosis, the consensus of surgical opinion would be that the localized injury precipitated the development of the disease in that area, although the joint may have been normal before the accident and the patient may have had active tuberculosis elsewhere in the lung or in a gland in his abdomen and would not have developed tuberculosis of the hip had he not sustained a contusion. In this type of case the quiescent period of six weeks is of great significance because it fits in with the present-day concept of how such disease develops and the rate at which it develops.

X-Ray Examination

The treatment of injuries arising out of and in the course of employment opens up a wide field for the radiologist. Probably in no other field of medicine does the clinician depend so much upon repeated X-ray examination as in Workmen's Compensation work. The opportunity for extensive and repeated use of X-rays imposes a great responsibility upon the radiolo-

gist. Careful and discriminating use of this method of investigation does a great deal towards the solution of many problems presented by injured persons, especially in those problems concerned with the relationship between pre-existing pathology and alleged injury. The X-rays are essential in diagnosis, in planning effective treatment, assessing disablement, analysing the causal relationship between the injury and the end result and, incidentally, for keeping an adequate record of all stages of the man's condition from accident to complete rehabilitation. It is not uncommon for X-ray examinations to be quite inadequate; for example a man may have a fractured thigh bone and the house surgeon may request an X-ray of the thigh bone only, whereas the man in fact may have a dislocated hip as well, which is not shown on the early pictures. A belated discovery of the dislocated hip as long as months afterwards is not only a possibility, but also a probability, in hospitals where the X-rays are not under the control of highly trained radiologists. In one hospital on one day I saw three Natives who had had fractures of thigh bones who were subsequently discovered to have dislocated hips when they refused to walk after the fractured femora were healed. Clear and adequate X-rays are therefore fundamental to early assessments and evaluation of disability. Throughout the period of treatment, there are pitfalls in such cases where adequate radiological examination can assist the clinician to do the best for the patient.

There is a great variety of conditions in which adequate radiological examination will produce evidence on which it can be determined whether the condition from which the man suffers had existed before the accident, or whether it developed as the result of the injury. The clinician requiring radiological examination of the patient indicates to the radiologist his tentative diagnosis and predicts the probable findings as far as possible to give the radiologist something concrete to work on, but in no way should he suggest to the radiologist to what extent the examination should be limited, although it may be correct for him to suggest one or two particular views that may be helpful. The ultimate responsibility for radiological diagnosis lies with the radiologist and if he decides to take fifty pictures of the cervical spine, it is entirely his own business. The question of expense in investigating a case such as this pales into insignificance in relation to the dangers of a mistaken diagnosis. It should be clearly understood, however, that the clinician is the man evaluating the disability and it is only by combining his clinical findings with the radiological report, that he is able to summarize comprehensively in his own mind the actual state of the victim of the accident. He should not be dominated by radiological opinion, but over a period of years will learn what value to attach to radiological opinions. The radiologist may report that the fracture is incompletely consolidated by bone, whereas the clinician may have no doubt in his own mind that at that stage it is already safe for the patient to bear a restricted amount of

weight on the healing fracture. If he had to wait until the radiologist was satisfied that the bone was really united, he would be rendering the patient a serious disservice and keeping him off work for months too long. The radiologist may discover a comparatively minor compression fracture of a lumbar vertebra. The clinician has to decide whether the man with the fractured vertebra can in fact get up, drive his car and go to work, even though the X-rays show a fresh fracture. Radiologists as a group are careful usually to keep within the bounds of their speciality, but they may unduly influence clinicians in decisions.

A radiologist may report that a fractured femur has a ½ inch of overlap, 1¼ inches of lateral displacement and 5° of forward angulation. This would look pretty unfavourable on paper or in a court of law, but the experienced surgeon dealing with fractures will know that if that fracture heals in that position, the man is likely to have perfect function of his thigh with a mobile hip and a mobile knee and with no discoverable disability, and that over-anxious endeavour to satisfy the radiologist that this fracture has been perfectly reduced, may end in much greater disaster than treating the man on conservative lines without too much interference. In assessing disablement, the functional end result matters most and X-rays shown in court may give very misleading impressions of the degree of disablement.

Malingering

In considering the problem of maligering, we are confronted by two directly opposed views. The diagnosis of malingering is usually made when it appears evident that the symptom might reasonably be conceived to attain ends for the patient which on an organic basis he would not be entitled to. By some method of clinical trickery in the course of examining such a patient, the doctor may demonstrate to his satisfaction that the particular symptom as demonstrated by the patient is assumed and not real. The tendency exists to regard such people as dishonest and deliberately deceiving the examiner. They are labelled lead-swingers, humbugs, malingerers and they are suspected of fraud. On the other hand, the present-day psychiatric attitude towards such a symptom is the direct opposite. The fact that the symptom is beneficial to the patient, or is calculated to bring about a substantial increase in the amount of compensation to be awarded in the settlement of his claim is, of itself, not an argument in favour of the symptom being that of a malingerer.

The normal physiological reactions of the body to disease, such as increased pulse rate, raised temperature, spasm of muscles to protect a painful joint, a drawn-up leg in acute appendicitis, are all beneficial reactions by the body to the disease and their distinct purpose is the furthering of the ends of the individual. No-one would regard such symptoms as being assumed. In the same way, the whole interplay of psychological

mechanisms is properly viewed from this standpoint. The hysterical patient who becomes an invalid to get an enormous amount of attention and to become the centre of interest and of authority in the household is a familiar picture. The neurotic who develops a paralysis or an anaesthesia of the hand or a leg, demonstrates symptoms calculated to advance his interests; yet I do not suppose anybody believes that these symptoms are simulated. The subtle gradation between an anxiety state, such as hysteria, and the state of the malingerer, is seen in every-day practice. If a patient has a paralysed arm, he may not be able to use it because of organic disease, and this is fairly readily detected by routine clinical examination. On the other hand, if he is simulating the paralysis, various tricks are customarily used to demonstrate that he is deceiving the examiner. For example, he might be handed a book too big to grasp with one hand, the examiner drops the book and the patient suddenly reflexly grasps after it with the paralysed as well as the normal hand. The so-called deception would have been uncovered. But the question is whether this man is malingering. The experiment has proved that the paralysis is not organic, i.e. it is psychogenic, but that may have been determined without exposing the patient to a test which now makes him look like a liar. The motivation of this man's behaviour is a matter for the psychiatrist. At this point the surgeon has to recognize the limitations of his own training. Throughout his period of training he has been taught to deal with organic disease, he has been taught to identify the paralysis of individual nerves, he has come to believe so strongly in his own clinical findings that he may be prepared to call this man a humbug and a fraud; but in the developments in psychiatry and psychology in recent years, it has become increasingly appreciated that the term malingering can only be applied in a very restricted way. It is only when the symptom can be demonstrated to have had its origin in a field of clear conscious awareness and that the patient had the conscious purpose in mind to deceive, to defraud, to bring about his own ends, that the term malingering could be used. It is not sufficient to label a man like this a malingerer and regard the matter as final and sufficiently assessed.

This simulation of disease is the individual's response to his real situation. His behaviour is an inadequate, inefficient way of dealing with reality. It appears to be the reaction of an inadequate personality, of a person who has to resort to lying, to drinking or to hysterical paralysis in his efforts to get along with every-day problems of reality which confront him. It must of necessity be conceived of as presenting a defective type of personality and no diagnosis of that individual is adequate, which picks out alone the obvious emphasized, superficial and prominent symptom of that defect and makes a diagnosis upon it alone. It may be true that the conscious motives make this man a malingerer, but the psychiatrist may

discover a perfectly plain defect of personality for which the symptoms would be plainly compensatory. The diagnosis of malingering cannot be made on the usefulness of the symptoms presented, because all symptoms are useful, although some are more obviously so than others.

It will be apparent that the gradation from a neurotic state with simulated symptoms merges with a state of conscious deception with simulated symptoms and it would be a bold clinician or perhaps an ignorant one who dares to make a diagnosis of liar, humbug, lead-swinger or malingerer. It is probable that patients who are plaintiffs in claims for compensation exaggerate their symptoms to a varying degree and that some will go far enough to be suspected of practising deception, but even this cannot be readily classified as conscious deliberate deception except in extreme cases, which must be very rare. When the patient practises deception deliberately he will ultimately believe his own story—as every fisherman knows.

A friend of mine while fishing in tropical waters, caught several fine fish. The circumstances were somewhat difficult, the weather threatening, the boat unseaworthy —in fact the background built him up to be a courageous, intrepid strong man, worthy of the respect of his neighbour. One of the Native crew of the boat caught a 135 lb. gerupa with a handline—the biggest fish of the day. By the time they got to the camp my friend was saying 'we caught the fish.' By the time we got to Johannesburg the fish and photographs were publicized and admired. The next year I saw a movie of the expedition and the angler said in the running commentary that he had had a terrific battle with the fish.

A surgeon of my acquaintance brought back the skin of a lion he had shot. He sometimes refers to it as his lion. But the facts are that four fatal bullets from four different rifles killed that lion and it could not be determined who fired the first shot. Perhaps possession of the skin determines the point.

Neither of these two gentlemen is accused of fraud. Allowance is made for their psychological aberrations. I would even say that by anglers' and hunters' standards they have been modest in staking their claims. In neither case did the fish or the lion increase in weight or dimensions in recent months.

Whether the so-called malingerer is after social prestige, or a claimant for human respect, or a martyr suffering torture or a plaintiff claiming money from a flourishing insurance carrier, makes little difference to the comprehensive clinical diagnosis which must include a psychological diagnosis.

In diagnosis of malingering the following factors are important:

1. The history of the previous condition, both medical and psychological. Information from acquaintances, fellow-workers or employers may be valuable.
2. Distraction of attention so that he may relax his grip on himself and on the act he is putting on.
3. Production of confession or bewilderment, e.g. by repeating tests of various sorts and demonstrating considerable inconsistency of response.
4. Malingerers have difficulty in reproducing some phenomena while under observation for days on end, e.g. disappearance of list of spine, limp, paralysis. Observation in hospital may be useful in such cases.

The examiner must be careful not to respond to suspected malingering by hostility of his own, or to over-sympathizing for one or other emotional reason.

Malingering and Anxiety States—Hysteria

By applying anatomy and physiology a certain number of cases can be elucidated. Sometimes by cajoling, persuading, bullying or bluffing, the malingerer can be trapped into committing a mistake.

Glove and Stocking Anaesthesia. If a person alleges that an injury has caused an anaesthesia or complete lack of sensation in a hand and halfway up the forearm, it is called a glove anaesthesia. If there is a clear-cut demarcation of the non-feeling zone distally and the feeling zone proximally, the phenomenon cannot be explained on the basis of a peripheral nerve injury. Nerves do not supply skin in such a way. The effects of a nerve injury are often remote from the lesion. Depending upon the branching of the nerve and the particular branches cut, certain standard effects are produced. These changes include sensory disturbances in specific areas and motor effects on certain muscles. Since most nerves have intermingling of motor and sensory fibres, isolation of sensory and motor effects by an injury is usually not possible. Knowledge of such detailed anatomy is necessary to trip up the humbug. Similarly, wrong combinations of sensory and motor disturbances are characteristic of hysteria.

Both the malingerer and the hysteric may say 'my hand is dead, I can't use it and I can't feel with it'. The hysterical patient is likely to exhibit a constant pattern of disability often for months on end. The malingerer, however, who has to mimic loss of function as a voluntary act, has an almost impossible performance to maintain day in and day out. He may relax, or get annoyed, or burn his finger or bounce away from an electrical or other noxious stimulus and so give the show away by using a hand which was supposed to be paralysed.

The yes-no test is often regarded as adequate proof of humbugging, but this need not necessarily be so. You will recall how it is done. The patient is blindfolded, the examiner gives clear-cut instructions: 'When you feel the pinprick say *Yes!* when you don't feel it say *No!*' After testing normal areas and getting a series of yesses he suddenly pricks the allegedly anaesthetic area which earlier was numb to pinprick and now gets a loud *No* as a response.

This test is more definitely of value when, after a few *No's*, the patient stops answering. He thinks he is being found out and shuts up altogether. By repeating the test he may be fooled into inconsistencies or he may become wiser and give answers which he regards as accurate.

At that stage the anatomical zoning becomes significant. If he shows an anaesthesia which cannot be explained on an organic basis, then he may

be a liar. However, certain conditions have to be fulfilled. If there has been demonstrable damage to the blood supply of a part, an anaesthesia or hypo-aesthesia resembling a glove or stocking anaesthesia may well occur. This glove anaesthesia is therefore due to humbugging or an anxiety state if the circulation is intact and not otherwise. This sort of test should be done before competent witnesses to increase its validity and the patient must be given a fair chance to understand instructions.

I have been able to prove this sort of thing to the presiding Commissioner at a Workmen's Compensation hearing, but it might fail if one attempted to repeat it in the august atmosphere of the Supreme Court. The expert witness might well ruin the value of his evidence while attempting to confound the plaintiff.

Briefly, the main distinguishing feature of the malingerer is that of the variability of his act from day to day or month to month. Consistency suggests psycho-neurosis. Inconsistency suggests humbugging. Sometimes hysterical cases are treated under pentothal analgesia by suggestion. The reponse to therapy is of diagnostic value.

If, as the result of suggestion, the patient gets better, he was probably only a hysterical case.

But malingerers tell their story so many times that they begin to live the part, to believe the story, and ultimately succeed in suggesting to themselves that they are genuinely disabled and then they become so in a psychoneurotic sense. Lead-swinging then fades into genuine hysteria. Only in the clear-cut cases would it be possible to be dogmatic.

If the man's performance is very consistent over a long period, he is probably hysterical.

Knowledge of Rehabilitation, Retraining and Vocational Training of Disabled Persons

In assessing disability the permanence of the disability has to be established.

If the person, although substantially physically disabled, is nevertheless capable of rehabilitation to varying degree, this must logically reduce his disability.

In other words, educability, motor ability, special talent in directions other than those involved in the accident, may minimize disability. This, of course, is a matter of which schedules in Workmen's Compensation Assessment take no cognizance.

This leads to the need for an estimate of the time required for rehabilitation or vocational retraining, the continued loss of earnings and the cost of the training and the status of the newly trained individual in a new labour market.

In this respect of an assessment expert knowledge in fields other than medicine have to be taken into account.

At an arbitrary point in such a consideration the doctor retires within the shell of his technical limitations, in favour of the vocational guidance officer, the aptitude testing psychologist and the placement officer from the office of the Director of Readjustment services.

References

1. White, W. A. (1919): *Simulation (Malingering) not an Adequate Diagnosis.* J. Nerv. Ment. Dis., **50,** 209.
2. Walshe, F. M. R. (1944): *Diseases of the Nervous System,* pp. 310–332. Edinburgh: E. & S. Livingstone, Ltd.
3. Kessler, H. H. (1931): *Accidental Injuries.* London: Henry Kimpton.
4. Brahdy and Kahn (1937): *Trauma and Disease.* London: Henry Kimpton.
5. Kessler, H. H. (1953): *Instructional Course Lectures of the American Academy of Orthopaedic Surgeons,* Vol. **XIII,** p. 292.
6. McBride, E. (1953): *Instructional Course Lectures of the American Academy of Orthopaedic Surgeons,* Vol. **X,** pp. 407–412.
   McBride, E. (1957): *Instructional Course Lectures of the American Academy of Orthopaedic Surgeons,* Vol. **XIV,** pp. 375–379.
7. McBride, E. (1953): *Disability Evaluation,* 5th ed. Philadelphia: J. B. Lippincott Co.
8. Coetsee, N. T., and Ungerer, V. (1947): *The Workmen's Compensation Act.* Pretoria: Union Booksellers (Pty.) Ltd.
9. Collie, J. (1933): *Workmen's Compensation: Its Medical Aspects.* London: Arnold.
10. Regan, Louis J. (1955): *American Academy of Orthopaedic Surgeons: The Medical Witness and his Testimony,* Vol. **XII,** pp. 269–274.

Identification of Menstrual Blood in Dried Stain Form

By an Electrophoretic Method

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The identification of menstrual blood and its differentiation from arterial and venous blood is a matter of considerable importance to the forensic scientist. For example, it is a common occurrence for a person accused of a crime of violence or of criminal abortion to explain blood stains on clothing as being due to menstrual blood innocently derived; while alternatively, a man accused of the rape of a victim who was menstruating at the time of the offence may explain the blood-stains on his clothing as due to a nose bleed or a cut finger. It is highly desirable that such statements should be capable of independent corroboration or contradiction.

The methods which have been used depend largely on the identification of vaginal debris. Taylor,[1] for example, lists 3 points of identification:

(*a*) Acid pH due to the admixture with vaginal mucus;

(*b*) Microscopic identification of vaginal epithelium;

(*c*) The presence of numerous micro-organisms.

Smith and Fiddes[2] emphasize the microscopic identification of endometrial cells.

All these methods depend on a microscopic examination and are not very satisfactory since only endometrial fragments, which cannot always be found, are specific for menstrual blood. All the other points of 'identification' are confirmatory rather than conclusive and all of these points could be due, in some circumstances, to contamination of venous blood.

Glaister[3] points out that no fibrin is visible in stained material. This is associated with the well-known fact that menstrual blood does not clot.

This lack of clotting is due to the fibrinolytic activity of menstrual blood.

Bell, Davidson and Scarborough[4] suggest that this activity is due to the lack of an inhibitor, antiplasmin, which is normally present in venous blood together with plasmin (a fibrinolysin). The antiplasmin normally prevents fibrinolytic activity.

The identification of this fibrinolytic activity as a delicate test for menstrual blood has already been suggested and methods based on the chemical determination of soluble protein products by micro-Kjeldahl nitrogen estimation have been described. Such methods, however, require meticulous technique and they are time consuming.

A simple method of detecting and demonstrating this fibrinolytic activity should be of material assistance in identifying menstrual blood. This

fibrinolytic activity converts insoluble fibrin into soluble fibrin, i.e. into soluble protein products capable of electrophoretic identification, a unique band due to these fibrinolytic products being produced in the electrophoretic pattern.

The Method

The heaviest part of the area of staining is cut from the cloth. For reasonably heavy staining a 1 cm. square is the maximum convenient. Very much smaller blood stains are normally used. The minimal quantity is a stain equivalent to about 2 drops of whole blood. The actual quantity varies from sample to sample.

The stained cloth is placed in a small (e.g. Wassermann) tube and distilled water is added. The quantity of water should be sufficient to allow thorough saturation of the cloth without any excess.

Due to the variation in the absorbency of different substrate materials it is impossible to give any fixed volume of water appropriate to a given size of stain.

The object in limiting the quantity of water necessary is to obtain the strongest extract possible, allowing for the absorbency of the material substrate and the fact that the quantity of final extract required should be a minimum of 60 microlitres. The maximum that is found convenient to handle at this stage is about 0.4 ml.

A similar blood stain of known non-menstrual origin is treated in the same way.

These 2 stains are allowed to soak for at least 20 minutes. The pieces of cloth should preferably be prodded from time to time to ensure good extraction of the stain.

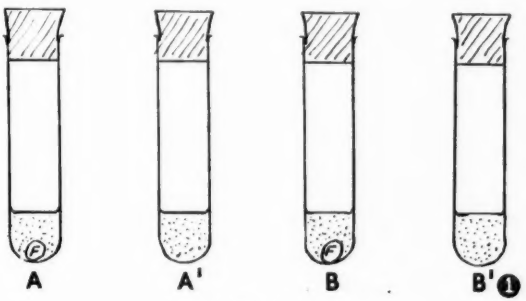

Fig. 1.

Non-Menstrual Blood Extract
A: With fibrin.
A¹: Without fibrin.

Suspected Blood Extract
B: With fibrin.
B¹: Without fibrin.

The cloth is then squeezed and the extract collected. Each extract, suspected menstrual and non-menstrual, is then divided into 2 equal parts. The minimum quantity of extract in each of the 4 tubes should now be 30 microlitres.

Fibrin is then added to one tube of each extract (suspected menstrual and known non-menstrual).

Various forms of fibrin have been tried—precipitated, dried foam, fresh and old fibrin obtained by defibrination.

The only one of these which gives satisfactory, consistent results is fresh fibrin obtained by defibrination as follows:

0.5–1.0 ml. of whole blood is defibrinated and the fibrin obtained is washed very thoroughly in saline (0.85) and then in distilled water.

This quantity of fibrin is then divided into 2 equal parts and one part is put into one tube of suspected menstrual extract and the other part is put into one tube of known non-menstrual extract.

The 4 tubes are placed in an incubator at 37° C. for 24 hours.

The electrophoresis tank is set up with a barbitone buffer of pH 8.6. The buffer used was as follows:

Sodium Barbitone: 33.0 g.
Sodium Acetate (hydrated): 19.5 g.
N/10 Hydrochloric Acid: 205 ml.
Water: to 3 litres.

The apparatus used was an LKB tank taking 4 strips of 4 cm. Whatman No. 1 paper in a horizontal position.

The current was supplied by a Shandon 'Vokam' power pack.

No other apparatus has been tried for this technique but there seems to be no reason why any other electrophoretic apparatus should not be used.

After the apparatus has been set up and reasonable time allowed for it to acquire stability, 20 microlitres of the extracts are applied, one to each paper.

The method of application is largely a matter of choice. I have personally preferred to use a capillary pipette, but a glass slide, brush, etc. may be used with equally satisfactory results.

The run is made overnight (about 16 hours) at 120–150 volts.

The strips are then dried at 105° C. for half an hour and stained with Azocarmine B.

The Result

The distinction of menstrual blood is apparent as an extra band due to fibrinolytic products on the paper strip on which the menstrual extract with added fibrin was placed.

No band of fibrinolytic products occurs in the cases of non-menstrual blood with or without fibrin or menstrual blood without fibrin (Fig. 2).

The degree of fibrinolysis varies from sample to sample and in some cases the extra band is weak and would probably be better estimated on a densitometer, but generally the result is apparent to the eye.

There would also appear to be a fall in fibrinolytic activity after the first day of menstruation, which can be very marked. This first day is, however the time of greatest flow, hence contamination is most likely to occur at this time.

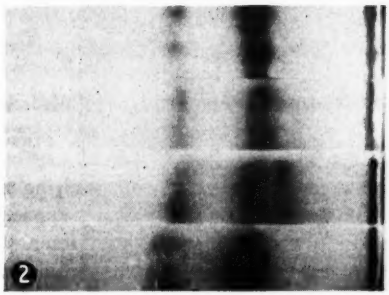

Fig. 2.

A: Capillary blood with fibrin.
B: Capillary blood without fibrin.
C: Menstrual blood with fibrin.
D: Menstrual blood without fibrin.

Summary

1. Microscopic methods for the identification of menstrual blood are mentioned.

2. A new method of identification based on the electrophoretic determination of fibrinolytic activity is described in detail.

References

1. Smith, S. and Simpson, K. (1956): *Taylor's Principles and Practice of Medical Jurisprudence*, 11th ed. London: J. and A. Churchill, Ltd.
2. Smith, S. and Fiddes, F. S. (1955): *Forensic Medicine*, 10th ed. London: J. and A. Churchill, Ltd.
3. Glaister, J. (1931): *Medical Jurisprudence and Toxicology*, 5th ed. Edinburgh: E. and S. Livingstone, Ltd.
4. Bell, G. H., Davidson, J. N. and Scarborough, H. (1953): *Textbook of Physiology and Biochemistry*, 2nd ed.

Pressure on the Neck

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During the past few years we have had an opportunity of studying a large number of cases in which death has been due to pressure applied to the neck. Many of the observations in this paper have been recorded elsewhere or may seem a matter of commonsense, but we have considered it worth correlating them in a single paper based upon these cases.

Although at first sight it appears somewhat elementary, it is essential, when discussing the effects of various types of trauma, to understand the significant anatomical arrangement of the structures and the physiological effects of pressure upon them. The relationships of the main structures of the neck to the larynx are shown in a series of diagrams and photographs of progressive dissection of the neck (Figs. 7, 9A and 9B).

Anatomy

The Hard Structures of the Larynx

The Hyoid Bone. Although this bone is relatively inaccessible, especially when the neck is flexed, its U-shape makes it vulnerable to fracture if the extremities of the greater cornua are forced together, e.g. when lateral compression takes place. The greater horn of the hyoid bone is united to the body of the bone by a plate of cartilage. When this plate is not ossified, considerable mobility may be present which the unwary may mistake for a fracture. The ossification of this cartilage is extremely variable and occasionally only one union is ossified. The lesser horns, which are united to the body by synovial joints, are rarely injured. The greater horns of the hyoid bone are overlapped by the sterno-mastoid muscle, the extent of the overlap depending on the position of the neck.

The Larynx. Below the hyoid bone the larynx is easily accessible to trauma, as it is separated from the surface in the median plane by only skin and two layers of fascia. The larynx lies in front of the bodies of the 4th, 5th and 6th cervical vertebrae. The upper border of the *thyroid*

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cartilage is in relation to the 5th cervical vertebra. Although this relationship is little changed by backward or forward movements of the head, its position with regard to the vertebrae is, however, altered when swallowing.

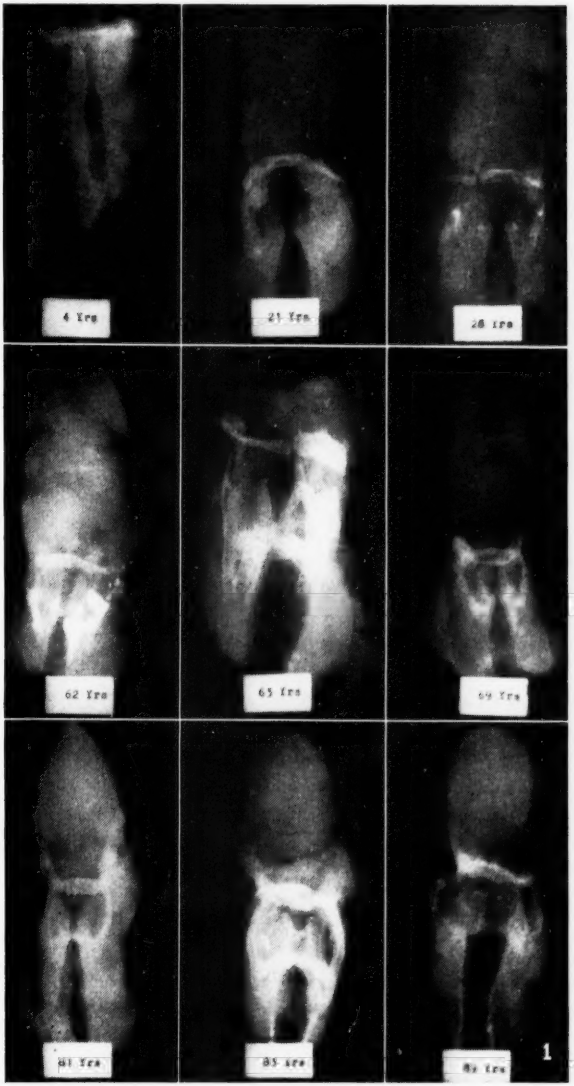


Fig. 1. Degree of calcification of thyroid cartilage is at various ages.

In the newly-born child the upper border is level with the body of the atlas, but after 4 years it reaches the adult position. *The superior horn* of the thyroid cartilage is strongly attached to the hyoid bone by the thyro-hyoid ligament, which may be an important mechanical contribution to the production of fractures of the superior cornu of the cartilage and to some fractures of the hyoid bone.

The V-shaped relationship of the two laminae of the thyroid cartilage in coronal section is maintained against a compressing force acting from the front only by the attachments of the inferior constrictor muscle forming the posterior pharyngeal wall, the synovial crico-thyroid joints and the crico-thyroid ligaments. The angle formed by the two laminae of the thyroid cartilage shows considerable variation and is said to be greater in the female, whilst in the infant the laminae meet in a curve rather than in the sharp angle of adult life. The amount of calcification of the thyroid cartilage is variable, and although it is commoner for calcification and ossification to increase with age, it is by no means unusual to find an almost completely cartilaginous thyroid cartilage in old age (Fig. 1). Just outside and behind the lateral borders of the thyroid cartilage are the carotid sheaths and in life, especially when the neck is not fully extended, there is considerable side-to-side mobility of the whole larynx; the carotid sheath also blends with the pre-tracheal fascia and moves to some extent with the larynx.

The Cricoid cartilage is the only firm structure which completely encircles the air passages. It may calcify but is commonly so elastic as to give the impression of being broken unless carefully dissected.

Venous Drainage of the Head and Neck

The venous drainage of the cranial cavity and of the head and neck is not only through the jugular system, but also through a series of intercommunicating veins related to the spinal column. These have been studied in detail by Batson,[3, 4] especially with respect to their role in the metastases of carcinoma. He has stated that the cross-sectional area of the external vertebral plexus in the neck is greater than the combined areas of the internal jugular vein. The vertebral venous system can be divided into:

1. The internal vertebral plexus;
2. The external vertebral plexus; and
3. The communicating veins.

1. *The Internal Vertebral Plexus.* This lies within the spinal canal, in man is probably limited in its blood carrying capacity but nevertheless is the only form of venous drainage of the cranial cavity which cannot be affected by any compression of the neck.

2. *The External Vertebral Plexus.* This is composed of a mass of thin-walled valveless veins in the deep muscles related to the spinal column and in the sub-occipital muscles.

As can be seen when both jugular veins are ligated during dissection of the neck for malignant disease, the vertebral venous system does not form an immediately available complete alternative route to the jugular veins for drainage of the blood from the head and neck. In most of the reported cases following ligature of both veins at the same operation there has been congestion and oedema of the face with oedema and suffusion of the conjunctivae. The disturbances following delayed ligation of the second jugular vein are less, and seldom fatal, presumably due to the opening up of the vertebral collateral circulation.[1, 8-10, 18] Poirier[13] mentioned that he had noted that in cases of hanging the face and neck were frequently congested although the brain appeared anaemic. He suggested that the intracerebral and vertebral venous plexus connexions allowed complete drainage of the brain. Batson has succeeded in filling the sub-papillary vessels of the face via the vertebral plexus with fluid injected into the dorsal vein of the penis of a cadaver, demonstrating that

Fig. 2. Rupture of post-cricoid venous plexus in death from coronary insufficiency.

the vertebral venous system might drain the rest of the face and head and neck despite compression of the jugular venous system. Gius and Grier[10] performed some interesting experiments on the bodies of babies stillborn or dying in the neo-natal period. After being suspended vertically until the blood had drained from the head, a tight compression bandage was put round the neck to occlude the jugular venous system and sodium iodide was injected into the superior longitudinal sinus. The vertebral plexus could then be seen radiographically as a continuous network of small channels extending along the lateral vertebral area down to the mediastinum where they entered the systemic veins.

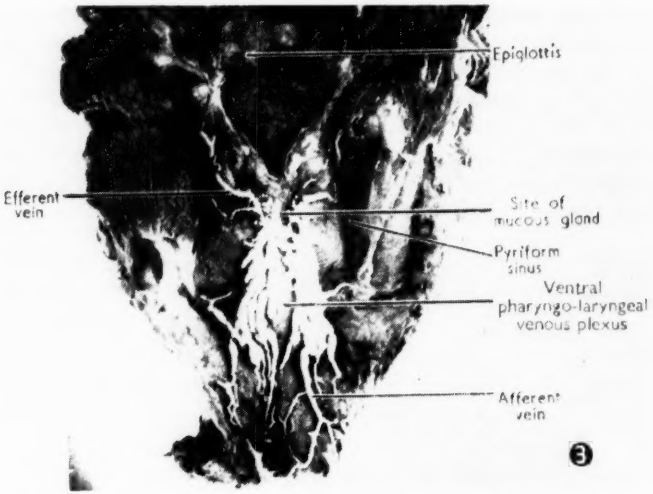


Fig. 3. Ventral pharyngo-laryngeal plexus (Courtesy of Prof. H. Butler, Department of Anatomy, Khartoum.)

Furthermore, in life, Batson[4] has shown that when opaque material is injected into the forearm veins with the breath held and especially when there is some straining against the closed glottis, the material will pass into the lower cervical vertebral plexus and return to the chest by this route rather than through the subclavian vein. He also suggests that the vertebral venous plexus is a normal pathway from the head and arms every time the breath is held forcibly, and that it may be better developed in those whose occupation or sport involves frequent breath-holding, such as in lifting and straining.

Venous Drainage of the Pharynx and Upper Oesophagus

This is of particular forensic interest because of the frequency of haemorrhage on the back of the larynx, usually over the dorsum of the cricoid

cartilage. These haemorrhages have commonly been interpreted as indicating direct pressure of the larynx on the spinal column, with subsequent bruising; in fact the majority are due to over-distension and rupture of a group of thin-walled venous sinuses forming the pharyngo-laryngeal plexus, and are no indication at all of local injury to the larynx (Fig. 2).

The upper oesophagus is drained by three groups of veins: the intrinsic and extrinsic veins and the venae comitantes of the vagus nerve.[6] The intrinsic group is further subdivided into subepithelial, submucous and perforating veins. The submucous group is the largest and drains into the pharyngo-laryngeal plexus. The pharyngo-laryngeal plexus is in two groups, the ventral group on the back of the cricoid cartilage, and the dorsal group in the posterior wall of the pharynx. The ventral group forms

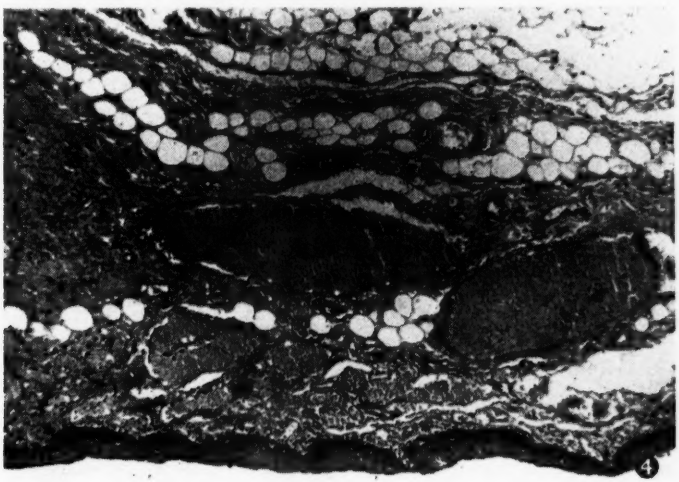


Fig. 4. Photomicrograph of a transverse section through pharyngolaryngeal plexus in Fig. 2. This demonstrates how thin-walled the plexus is in life.

two longitudinal masses on either side of the midline of the dorsa of the cricoid, separated by a gap varying from 2 to 6 mm. The individual veins forming the pharyngo-laryngeal plexus are slightly tortuous and show fusiform and globular dilatations up to 4 mm. in diameter in the adult (Fig. 3). Because most bodies are placed in the supine position after death, the veins are compressed by the weight of the larynx and may not be obvious. This varicosity is seen at all ages and, together with the thinness of the walls, partly accounts for the frequency of rupture (Fig. 4). The plexus drains into the superficial pharyngeal veins, and in so doing the efferents from the pharyngo-laryngeal group all have to

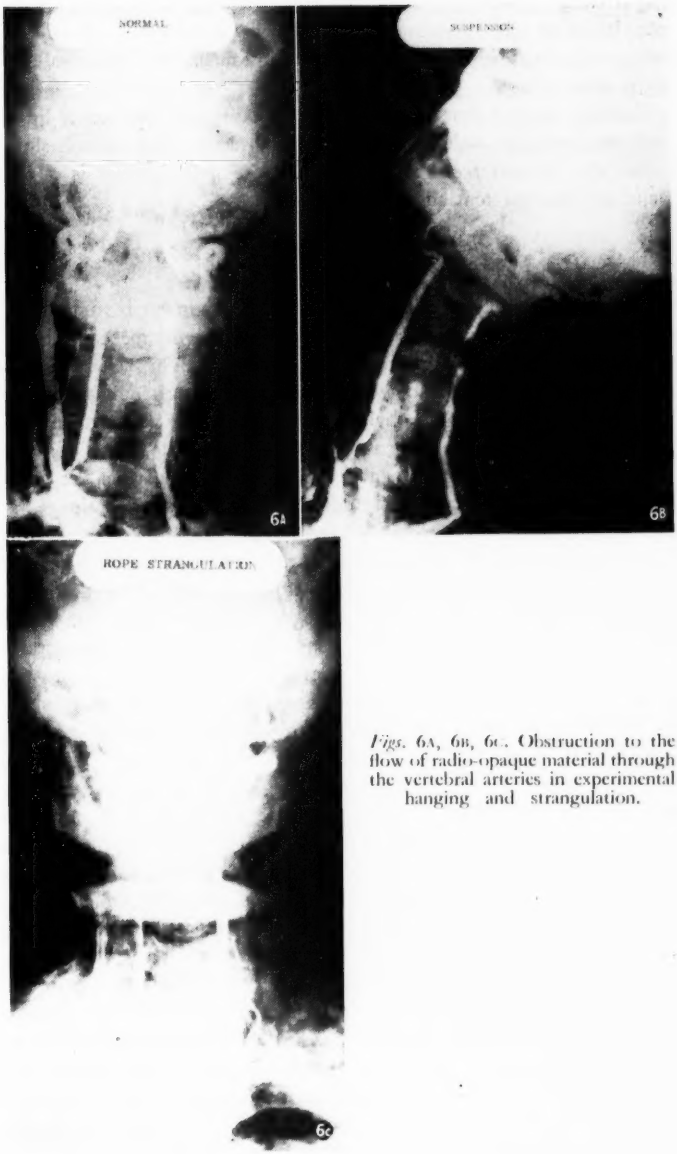
pass through the muscular wall of the pharynx, and hence may be particularly vulnerable to obstruction during muscular contraction such as might occur in strangulation. Occasionally thrombosis occurs, especially in association with congestive death (Fig. 5).

Fig. 5. Thrombosis in the pharyngo-laryngeal plexus.

The Arterial Supply

The common carotid arteries above the level of the cricoid cartilage are separated from the surface of the neck in an antero-lateral direction by only the sterno-mastoid muscle, the deep cervical fascia, platysma and skin. Posteriorly they rest on the firm surface provided by the transverse processes of the 4th, 5th and 6th cervical vertebrae and the longus cervicis and capitis muscles. The common carotids are therefore liable to compression by direct pressure, especially where they cross the transverse process of the 6th cervical vertebrae.

The vertebral artery, on the other hand, is probably rarely affected by direct pressure, although in some cadavers we have found that the free flow of radio-opaque material through the vertebral arteries can be



Figs. 6A, 6B, 6C. Obstruction to the flow of radio-opaque material through the vertebral arteries in experimental hanging and strangulation.

obstructed by extremes of rotation and lateral flexion of the neck, such as might occur in hanging (Figs. 6A-6C). The amount of blood carried to the brain by the vertebral arteries varies considerably in different subjects, and consequently the ease of establishment of a collateral circulation is also variable.

Brouardel[5] claimed that a tension of 30 kg. is necessary on a ligature around the neck to occlude the vertebral arteries. Rauschke[16] showed that the least tension required to block all the neck arteries is 5 to 10 kg., and the greatest tension is 20 to 30 kg.

Physiology

It is difficult to apply the findings of the experimental study of the physiology of anoxia to the practical problem of the mechanical effect of the various types of pressure to be discussed. The structures compressed or damaged and the degree of compression may only be assessed with difficulty and they must obviously vary from case to case. Suicidal, homicidal and accidental happenings do not have the same controlled conditions as laboratory experiments; during part of the time the arterial supply may be affected, during another part the venous drainage, and during another part, the airway.

However, the work of Swann and Brucer[19] is of interest. They have described the cardio-respiratory and biochemical events during death from obstructive asphyxia in unanaesthetized dogs. The obstruction was brought about by closed face masks. The dogs survived about 8 minutes in contrast to the 5-minute survival of dogs breathing pure nitrogen. Contrary to the generally accepted view, they found that the heart slowed considerably within 90 seconds of the obstruction. It remained slow except for terminal bouts of tachycardia, and the slow rate was accompanied by a great elevation of systolic, and a fall of diastolic, pressure. Heart failure occurred abruptly. Ventricular fibrillation did not occur. Arterial oxygen saturations fell progressively. The CO_2 content first rose rapidly, but when the blood lactate began to increase, the CO_2 content fell. The CO_2 tension rose steadily, the blood pH fell throughout and finally there was extreme acidosis. Breathing movements continued strongly until the terminal cardiac failure occurred.

The time taken for unconsciousness, and later for death, to arise is extremely variable, presumably because of the number of factors involved. Furthermore, it may be that reflex cardiac arrest can supervene at any stage.

It has been recognized for a considerable time that a nervous stimulus can cause rapid death, and Casper[7] stated that death from suffocation could happen rapidly from 'nervous apoplexy' in which there was no trace discoverable by the anatomist's knife. This form of reflex, commonly called 'vagal inhibition,' which we prefer to call 'reflex cardiac arrest,' is probably

initiated by alteration in carotid sinus pressure, the vagus nerve trunk in man being comparatively inexcitable to mechanical stimulation. Carotid sinus stimulation in man can produce effects varying from transient cardiac slowing, hypotension and vasodilation, to syncope, cardiac arrest and possibly death. Very marked cardiac slowing can be produced in arteriosclerotic subjects, but unfortunately there are no records available to show whether strangulation in older people with carotid artery atheroma produces more rapid death.

The Effects of Pressure on the Neck

If death follows pressure on the neck there are three questions to be answered:

1. How was it applied?
2. How much force was used?
3. For how long was the force maintained?

In many cases the third consideration assumes a major importance. It is often said that because death was rapid it was due to 'vagal inhibition,' and because 'vagal inhibition' has been known to result from minor trauma, a particular death was due to minor trauma, and was not murder. This somewhat illogical medical reasoning ignores the legal fact that the most important part of the proof of murder is the proof of intent. This is indicated in the medical findings by the manner and force of application, rather than by how long the person took to die. The length of time during which pressure was applied is obviously relevant, for deliberate manual compression for 5 minutes can scarcely be without intent, even if the person died when the hands touched the throat.

Since we consider that the medical witness should give his opinion in these terms we will consider each question separately.

The Manner of Application (Local Injuries)

1. *Evidence from the Skin.* If the pressure has been applied by the fingers there may be abrasions from the finger nails or bruising from the fingers. These will be located according to the method of application and it is clear that considerable variation may be expected and hence each case must be interpreted on a basis of common sense. The appearance of nail scratches, for example, will obviously depend on the length and contour of the assailant's finger nails, and their presence upon whether there are protruding finger nails at all (many assailants are nail biters). Irregularities in the edges of the finger nails give characteristic marks. Bruising will be greater if the grip has shifted or been reapplied, or if the victim struggles. If the pressure is maintained in one spot until the victim's circulation ceases, it is possible that no bruising will be seen because there has been no blood flowing to extravasate from the damaged

vessels. It must not be forgotten that the victim may scratch and mark his own neck in an endeavour to free himself of attack, and therefore the presence of fingernail scratches on the neck does not necessarily mean that strangulation was manual. The direction of the mark may then be of considerable significance.

The effects of a ligature on the skin depend on the roughness of the ligature, its mode of application and the degree of movement. The typical roughened mark of rope used for suicidal hanging, which becomes dried and brown after death, is a good example. This appearance, which is due to friction, does not necessarily indicate a vital change, and we have produced similar marks by suspension of a cadaver. A smoother ligature, such as a nylon stocking, may leave very little or no mark, especially if removed shortly after application. In all cases it is worth examining the suspect marks by low power magnification with an oblique light, as there may be a pattern from the cord or cloth used as a ligature. Examination of the mark may also indicate the situation of a knot or loop. If the ligature has encircled the neck twice, frequently the skin between the two loops is pinched and haemorrhagic.

The presence of petechiae immediately above a mark and their absence below it is strong evidence that this mark is an indication of compression of the neck. This may be confirmed by a vertical histological section from above to below the line of compression to show congestion and rupture of capillaries above. This observation may be of value especially in cases of alleged strangulation of new-born children. A soft piece of cloth is frequently used, being the nearest available material to the mother. The marks produced are often negligible or absent and furthermore the skin of a baby's neck is usually creased and furrowed, making the determination of a ligature mark even more difficult.

2. *Evidence from the Subcutaneous and Soft Tissues of the Neck.* In manual strangulation there are usually localized haemorrhages in the subcutaneous tissues and the muscles of the neck, corresponding to the position of the fingers. Their distribution is of the greatest importance. Several areas of bruising at different levels is good evidence of pressure from fingers rather than from a ligature. If the bruising is from a ligature it will usually be at a similar level. Bruising from violent blows on the neck is usually more severe and widespread than bruising from finger pressure, but the single blow on the front of the neck which may cause immediate death may leave little trace.

Bruising does not invariably result from a ligature applied in life, especially when the ligature has been tightly secured and not removed until after circulation has ceased. This applies particularly to the effects of the ligature in many cases of suicidal hanging, from which there are frequently few, if any, vital local changes.

3. *Evidence from the Larynx*: (*a*) *The Hyoid Bone.* It is sometimes said and frequently suggested, especially in textbooks of forensic medicine, that fracture of this bone is specific of manual strangulation. Observations lead us to believe that this is far too dogmatic a statement. We have seen such fractures as a result of direct trauma, of suspension and even of post-mortem artefact.

It seems likely that fracture of the hyoid bone may be of two sorts: from direct lateral compression and by indirect violence. *Direct lateral compression* is probably the commonest mechanism in manual strangulation and, in order to produce this, pressure must be applied up under the angles of the jaw. It can be reproduced fairly easily in the cadaver, although considerable force is usually necessary and both greater cornua have to be pressed together.

Fig. 7. Mechanism of indirect fracture of the hyoid bone.

The second mechanism, *indirect violence,* is illustrated in Fig. 7. The hyoid bone is drawn upwards and immobilized very rigidly by the mass of muscles attached to its upper and anterior surface when the neck muscles are contracted, e.g. in an attempt to resist strangulation. Violent downward or lateral movement of the thyroid cartilage will exert traction through the thyro-hyoid membrane and especially the thyro-hyoid ligament and cause a traction fracture of the hyoid bone. The evidence that this second mechanism occurs can only be indirect, as it cannot be reproduced in the cadaver, for after death the hyoid bone is no longer capable of being firmly immo-

bilized. This may be the reason for Casper's statement that fractures of the larynx cannot be produced after death. However, the comparative frequency of fractures of both the greater cornu of the thyroid cartilage and the hyoid bone in hanging with an open loop (here our experience differs from that of Polson[14] who considers this rare), as well as in some cases of manual strangulation, is in favour of the existence of this mechanism. Further, when the thyroid cartilage is thus manipulated in dissection of the neck, the hyoid bone can be seen to be pulled upon.

(*b*) *The Thyroid Cartilage.* Injuries may be divided into fractures of the superior cornu and fractures of the body. The *superior cornu fractures* are the most common. They may be unilateral or bilateral, and are due either to the mechanism already described (i.e. from traction on the thyrohyoid ligament) or possibly from direct compression injury against the cervical spine.

Fractures of the body in the midline between two laminae are very rare, and we have only seen them from direct blows on to the laryngeal prominence, in one case by the blow from an assailant (R. *v.* Emmett-Dunne,

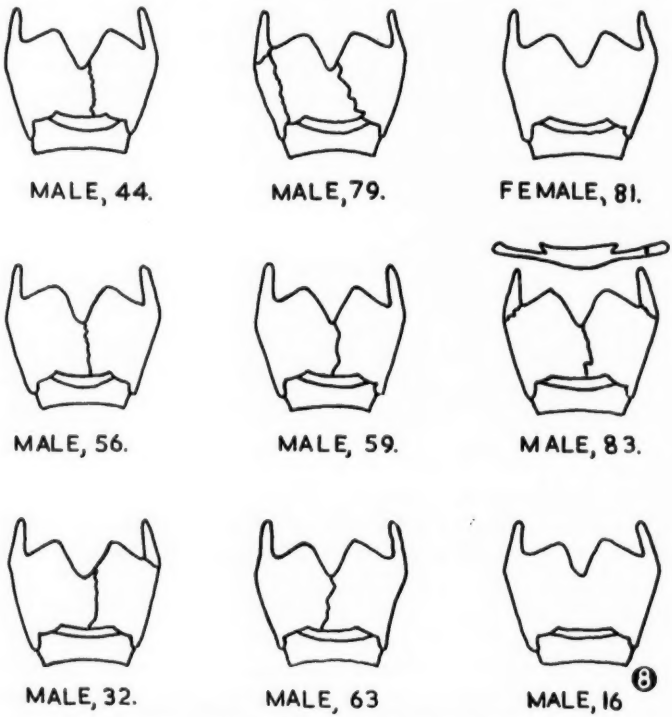


Fig. 8. Sites of experimental fractures of the larynx from direct blows.

1955), and in other cases from a blow against the handle bars of a motorcycle, the instrument panel of a car and a fall on to the top of a narrow brick wall.

Experimentally, the production of such a fracture depends upon some degree of calcification of the laminae, for when they are still cartilaginous fracture does not occur. There is considerable variation which is not entirely dependent upon age. The sites of fractures from such blows is usually slightly to one side of the prominence and in a vertical plane (Fig. 8). It appears that a blow of this type may cause sudden death attributable to trauma to the carotid sinuses (Figs. 9A, 9B) or reflexly from the laryngeal stimulus. It may not, however, always be immediate but after a short period which enables pulmonary oedema to develop (Muller, personal communication). Fracture of the thyroid cartilage, if survival takes place, may be followed later by adhesive laryngitis with obstruction.

We have also seen a fracture of the superior thyroid cornu in a man dying of ascending pyelo-nephritis in a hospital ward. There was no question of any ante-mortem violence, but the laying-out of the body was admitted to have been done very roughly with hyperextension of the neck. Furthermore, there was a localized post-mortem extravasation of blood around the fracture. This would, in less certain circumstances, undoubtedly have been considered good evidence that the injury occurred before death, and illustrates the care required in interpreting such injuries.

(*c*) *The Cricoid Cartilage.* In our series there are only two cases with fractures of the cricoid cartilage. One was due to a direct blow on the laryngeal prominence from an assailant and was associated with a midline fracture of the thyroid cartilage. The other was due to compression of the neck, probably applied with the assailant's forearm, with the assailant standing behind the victim. In this case the fracture was at the junction of the lamina and the arch and was associated with a fracture of the inferior cornu of the thyroid cartilage on the other side.

4. *Evidence from the Cervical Spine.* This is usually only injured in hanging with a long drop, or from violence other than pressure on the neck, and is therefore not relevant in this context. Post-mortem fractures of the cervical spine are not uncommon and may be caused by forcible 'laying-out,' by violence in moving the body and other unduly rough manipulation of the head, especially in forced flexion in reflecting the scalp. These fractures are usually transverse and related to the intervertebral discs between C4 and 5, or C5 and 6. A small extravasation of blood into the pre-vertebral fascia related to these fractures may also occur as a post-mortem artefact. They are especially liable to occur when ankylosis is present.

5. *Evidence from the Carotid Arteries.* Transverse intimal tears just below the bifurcation of the common cartoid arteries are occasionally seen in hanging even with a short drop. They are very frequently present after

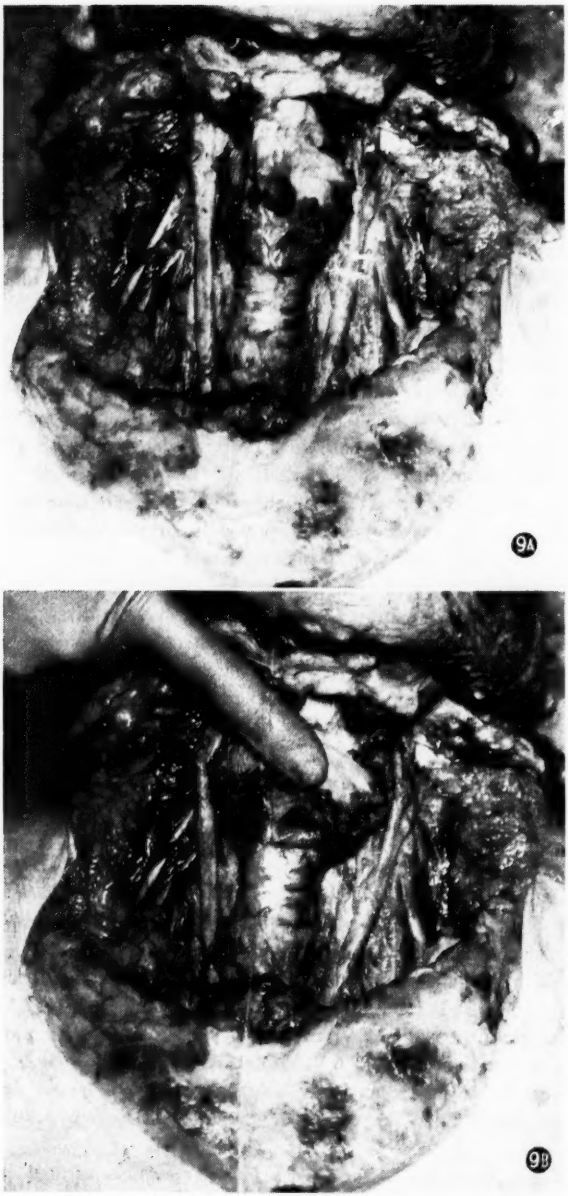


Fig. 9. Possible mechanism of trauma to carotid sinuses from a direct blow on the larynx.

death from judicial hanging. Longitudinal tears were described by the pathologist who did the original autopsy in the Emmett-Dunne case, when death followed a direct blow on the front of the neck. The only explanation appears to be a tear of direct injury either from the blow itself or from the arteries being struck by the laminae of the thyroid cartilage (as in Figs. 9A—9B). Intimal tearing with subsequent medial dissection has been described from a blow by a rope trailing from a motor vehicle,[12] and by a perforating injury of the pharynx (Derrick, personal communication).

The Length of Time of Application

Clearly it is only possible to attempt to judge from autopsy findings the length of time between application and death, for after the circulation has ceased, so too will the production of gross vital changes cease. In suspension this is self-evident but in manual strangulation death may also occur very rapidly although the murderous grip is maintained for a much longer period. In one case a man admitted squeezing the throat of his wife for 5 minutes, although the post-mortem findings suggested that she died when the pressure was first applied (R. *vs.* Pye, Essex Assizes, 1952).

It is clear, therefore, that any conclusions on the length of the application must be based on two considerations:

1. The general picture of so-called ' asphyxia.'
2. The extent of local vital reaction to the agent causing the pressure.

From these considerations we recognize two distinct extremes of post-mortem appearance due to pressure on the neck, which we have for convenience labelled ' Class I ' and ' Class II,' although there are all gradations between the two classes.

Class I. These are cases showing well developed skin, conjunctival and visceral petechiae, oedema of the conjunctivae, pulmonary oedema and a haemorrhagic response to the local injury of the neck, and well marked congestion of the root of the tongue. These must be accompanied by evidence of compression, for without this all these findings must be interpreted with considerable caution, for the following reason:

Venous congestion alone is so common a finding in death of any sort that little importance can be placed on it. Again, capillary congestion of the face can be found in many causes of death, and can also be caused by hypostases after death. Even some hours after death congestion of the face can on occasion be produced in a matter of seconds by change of position, as for example when the body is turned on its face for an examination of the back. A cadaver which has been lying for some time face downwards, or with the head dependent, may show oedema of the conjunctivae as a post-mortem phenomenon, and coarse petechiae may appear in the areas of hypostases, as a similar manifestation. Cyanosis (lividity)—correctly described by Shapiro[17] as a clinical observation, is,

when observed after death, probably the most fallacious and dangerous finding on which to base an opinion. Bodies dead from a variety of causes, including instantaneous death from injury, may be markedly 'cyanosed,' and the 'cyanosis' may appear in the post-mortem interval. Post-mortem trauma in a congested and 'cyanosed' body may lead to extravasation of blood. Although the presence of oedema of the lungs may on some occasions be a reliable guide to the fact that death has not taken place immediately, great caution must be exercised, as post-mortem oedema of the posterior (or dependent) parts may be confused by the inexperienced dissector with true oedema. Sub-pleural blebs of interstitial emphysema are sometimes found when violent expiratory movements have been made against an obstruction, but this must be interpreted with the knowledge that very similar blebs can be due to post-mortem decomposition.

It seems likely that in some circumstances visceral petechiae can appear after death (Hunt;[11] Gordon *et al.*[20]), and they are such a common finding in so many different types of death from all causes that any interpretation of their significance is open to question. Petechiae in the brain substance are sometimes said to occur in asphyxia. We have never yet seen such cerebral petechiae in asphyxia, and can only suggest with great trepidation that the petechiae described by these authorities are in fact the cut ends of congested vessels.

It is in the head and neck above the level of compression that the recognition of congestive changes is of vital importance. If the Class I changes are very prominent, it means that sufficient pressure has been applied to obstruct the venous return, but not the arterial supply. This appearance has been best exemplified in all 5 cases of self-strangulation we have had the opportunity of studying (Fig. 10), for they showed uniformly the most marked Class I changes that we have ever seen, and for this reason the following description of a case is included:

> An elderly woman was found dead lying across her bed. The room was locked on the inside and any possibility of outside interference could be excluded. Around her neck were tied two separate pieces of black elastic. The face and neck above the constricting ligatures was blue and swollen. Small haemorrhages were present in the skin above the ligatures. Further haemorrhages were found in the conjunctivae (which showed oedema), eyelids, mucosal surfaces and scalp. The lymphoid tissue at the root of the tongue was so congested and studded with haemorrhages as to give the impression of bruising, whilst there were many petechiae in the epiglottis and larynx above the ligature. The viscera presented the appearance of a congestive death.

It appears that self-application of a ligature can be carried out with enough force to prevent venous drainage through both the vertebral and the jugular systems. Although this degree of congestion can be produced by homicidal strangulation by ligature, in our experience this is rare, probably because an assailant uses excessive force. Usually in such cases the Class I changes are not so well marked. There are probably two further reasons for this: firstly, with a struggling victim it is more difficult to occlude the vertebral venous system and, secondly, after a murderous assault

the strangling agent is often removed when the victim dies, and consequently blood is allowed to drain passively from the head after death, thus lessening the degree of capillary congestion. In our experience the absence of such a gross picture excludes self-strangulation.

It is impossible to give a definite time during which the pressure has to be applied to produce various degrees of Class I changes, but as a general rule it seems that if the changes are well developed the pressure has usually been maintained for at least half a minute and probably longer. In saying this, it should be pointed out that few people realize how long half a minute is when timed.

Fig. 10. Self strangulation, showing congestion and haemorrhage at the root of the tongue and in the larynx.

Class II. These are the cases in which there are none of the aforementioned changes; the head and neck are pale, there are no skin or conjunctival petechiae and there is no pulmonary oedema. This appearance, or rather lack of appearance, can be brought about in two ways:

1. Death has been so rapid that insufficient time has elapsed for congestive changes to appear.
2. The force applied has been great enough to occlude the arteries to the head and neck.

The first group includes those cases in which one has to accept that death has probably occurred from sudden cardiac arrest. The second

group comprises many cases of suicidal hanging. It is difficult, however, to exclude cases of suicidal hanging from group I.

The Force of Application

If there is well developed localized bruising of the skin and soft tissues (not to be confused with extravasation from pressure), or if the larynx is grossly injured, this is undoubtedly evidence of considerable violence. Unfortunately, if severe force has been used, and death has been very rapid, there may be no time for bruising to develop and, unless the larynx is injured (which will depend to some extent on calcification), it may be impossible to determine how much violence was used.

There can be no doubt that manual strangulation with considerable violence can occur without laryngeal injury. Some indication of the type of pressure and of the force used is given by the degree of development of the congestive changes described. If they are well marked, obviously considerable force has been applied to and maintained on the neck and has involved sufficient of the circumference of the neck to occlude a significant proportion of the venous channels, both jugular and vertebral. Such a compression need not be fatal and it must not be assumed that the pressure which has produced the congestion is necessarily the same as that which has produced injury to the larynx; they may be independent phenomena. Victims of attempted strangulation may well show petechial haemorrhages in their eyes for some time after the assault. So, too, can persons who have had severe paroxysms of coughing or severe vomiting.

Appendix

Dissection of the Neck

Prinsloo and Gordon[15] pointed out the importance of careful dissection of the neck. They show illustrations and photomicrographs of dissected artefacts quite indistinguishable from ante-mortem bruises. We have had numerous similar experiences ourselves, especially in the dissection of the neck shortly after death. Friedman (personal communication) recommends prior removal of the brain and division of the trachea below the level of the sternal notch and removal of the chest organs before even removing the skin from the neck. This technique drains the neck veins of blood and undoubtedly reduces the incidence of dissection artefacts. We have obtained similar results by removal of the brain and incision of the superior vena cava without going to the length of dividing the trachea, which may alter the position of foreign bodies or tracheal secretion.

The skin of the neck should be reflected either by a V-shaped incision extending on each side from behind the ear to the angle of the sternum, or by a midline incision with extensions along the lower borders of the mandible.

Each muscle is divided and reflected individually and the front of the thyroid cartilage and the hyoid bone laid bare before removal. Removal is then performed with the utmost care, and after fixation the larynx is radiographed before further dissection.

The bodies of the vertebral and cervical discs may be displayed with a large chisel in a search for fracture-dislocation or haemorrhage into the disc spaces, and the spinal cord removed by a posterior approach. If any lesion of the spine is suspected, however, the whole cervical column should be removed and radiographed before dissection.

Summary

Some aspects of the normal anatomy of the neck are described and the interpretation of the results of pressure on the neck discussed.

Of particular importance to forensic pathologists is the recognition of the changes due to congestion of the head and neck from venous obstruction during strangulation, and differentiation from other appearances of a more innocent character. For, unless the dissector recognizes the limitations, a miscarriage of justice might well occur.

We should like to express our appreciation to all the coroners who have allowed us to quote their cases and who have helped us in many ways, especially Sir Bentley Purchase and Dr. R. I. Milne; also to Professor Richard Harrison, Department of Anatomy of the London Hospital.

References

1. Baldwin, J. F. (1902): Ann. Surg., **36,** 394.
2. Batson, O. V. (1942): Ann. Int. Med., **16,** 38.
3. Batson, O. V. (1944): Fed. Proc., **3,** 139.
4. Batson, O. V. (1950): Surgery, **28,** 320.
5. Brouardel, P. (1897): *La Pendaison.* Paris: Baillière.
6. Butler, H. (1951): Thorax, **6,** 276.
7. Casper, J. L. (1862): Casper's *Forensic Medicine,* Vol. II. London: The New Sydenham Society.
8. Dangel, M. (1905): Beit. Z. Klin. Chir., **46,** 495.
9. Evans, M. C. (1942): Ann. Otol. Rhin. Laryng., **51,** 615.
10. Gius, J. A. and Grier, D. H. (1950): Surgery, **28,** 305.
11. Hunt, A. C. (1958): J. Forensic Med., **5,** 141.
12. Northcroft, G. B. and Morgan, A. D. (1944): Brit. J. Surg., **32,** 105.
13. Poirier, P. and Charpy, A. (1914): *Traité d'Anatomie Humaine.* Paris: Masson.
14. Polson, C. J. (1955): *The Essentials of Forensic Medicine.* London: English University Press.
15. Prinsloo, I. and Gordon, I. (1951): S. Afr. Med. J., **25,** 358.
16. Rauschke, J. (1957): Deutsche Ztschr. Ges. Gerichtl. Med., **46,** 206.
17. Shapiro, H. A. (1954): J. Forensic Med., **1,** 144.
18. Sugarbaker, E. P. and Wiley, H. M. (1951): Cancer, **4,** 242.
19. Swann, H. G. and Brucer, M. (1949): Texas Rep. Biol. Med., **7,** 511.
20. Gordon, I. *et al.* (1955): J. Forensic Med., **2,** 31.

Survival after a Penetrating Injury of the Heart

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It is a general belief that penetrating injuries of the heart are necessarily fatal, and that death occurs within a very short time, if not instantaneously. In a number of cases reported, however, death did not occur. In the present case, the patient recovered after a penetrating wound into the right ventricle. The site of the external injury was such that it appeared unlikely that the heart would be injured without injury to the lungs.

M. D. S., *aet.* 35, was admitted to the General Hospital, Colombo, at 4.10 p.m. on 1 January 1958, with a history of a stab wound of the left side of the chest, inflicted at about 3 p.m. the same day. He was conscious and restless, with cold and clammy extremities. He was very pale, his pulse rate was 120 per minute, and the volume and tension were low. The blood pressure was 70/50 mm. Hg, and the respiratory rate was 66 per minute. Externally, there was a vertical stab wound on the left side of the chest through the 4th intercostal space, in the mid-axillary line and 1 inch long. Surgical emphysema was present around the wound and there was a certain amount of bleeding. The apex beat of the heart could not be felt, and the heart sounds were faint. The trachea showed no obvious deviation, the left chest was dull to percussion and the breath sounds were inaudible.

Intravenous infusion of Dextraven was started immediately, while the blood transfusion was being prepared. A watch was kept on the rising pulse rate; at 9.30 p.m. it was 134 per minute. At this stage the condition was assessed as one of left sided haemothorax with continuing bleeding, probably from injury to either an intercostal vessel or the upper lobe of the left lung. X-ray showed almost complete opacity of the left side of the chest with an area of compressed lung tissue at the apex (Fig. 1). As the haemorrhage seemed to continue unabated, and as there was no sign of any response to the rescuscitation measures in progress, it was decided to do a thoracotomy on the patient.

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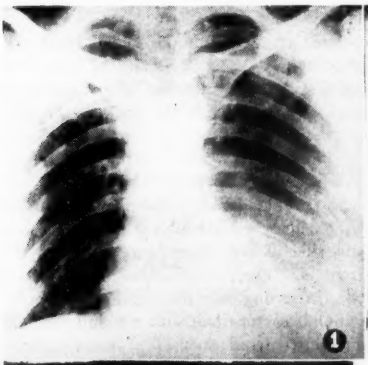
He submitted to surgery at 10.30 p.m. the same night, under nitrous oxide and oxygen, losing consciousness in about 3 minutes. Relaxation of the vocal cords was induced by succinyl choline hydrochloride (Scoline) 100 mg. intravenously. The succeeding respiratory arrest was treated by forced ventilation with pure oxygen,



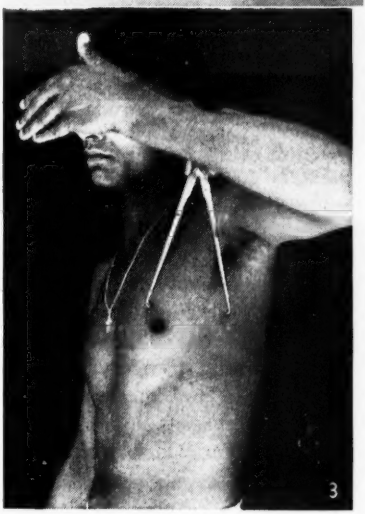


Fig. 1. Pre-operative X-ray of the chest showing massive haemothorax.

Fig. 2. X-ray taken 36 hours after operation showing lung fields and pericardium clear of effusion.

Fig. 3. Estimation of distance traversed by the knife, by external measurements of the scar visible on the left side of the chest in the mid-axillary line.

using the face mask and the re-breathing bag. Tracheal intubation was carried out with a size 8 cuffed endotracheal tube under direct vision. The cuff of the tube was inflated to make an airtight connexion between the trachea and the anaesthetic apparatus, and anaesthesia was maintained with nitrous oxide and oxygen in the proportion of 4:3, respiration being controlled manually. When the effect of the Scoline was passing off, 25 mg. of d-tubocurarine hydrochloride (Tubarine) for further relaxation, and pethidine 50 mg. with Atropine gr. 1/100, were given intravenously. Galamine triethiodide (Flaxedil) which is known to cause tachycardia, was avoided. Carbon dioxide accumulation (which increases cardiac irritability and

causes extrasystoles and arrhythmias which may lead to tachycardia or ventricular fibrillation) had to be carefully avoided throughout the operation. The respiratory movements were deliberately stopped to limit the movements of the chest wall and the diaphragm during the operation. No other anaesthetics were used, reliance being placed entirely on the nitrous oxide and oxygen with 2 further doses of Tubarine 10 mg. when the relaxation appeared to be passing off.

An oblique incision was made over the sixth left rib from the lateral border of the scapula forwards to almost the sternal margin. About 8 inches of the sixth rib were resected and the pleura opened. The extensive collection of intra-thoracic blood was evacuated, and the space inspected. No injury to the lung was found. Retraction of the lung revealed a penetrating wound in the pericardium 1 inch long, from which there was a continuous oozing of blood. A further 2 inches of rib were resected anteriorly, and retraction provided adequate space to work in. The pericardial sac was then opened further and a large blood clot inside the pericardial cavity was removed revealing a wound in the lateral surface of the right ventricle ¾ inch long. This was situated on the right side of the descending branch of the left coronary artery about ⅛ inch away from it. Every contraction of the heart resulted in a spurt of blood from this incision, showing that the incision had penetrated into the ventricular cavity. This incision was repaired with chromic No. 00 catgut and the pericardium was closed with interrupted stitches of catgut placed 1 inch apart, to provide exit for any further bleeding and avoid further cardiac tamponade. The anaesthetist then expanded the lung fully, after which it was inspected again. The pleural cavity was drained to a water-sealed bottle by an incision in a lower intercostal space, and the wound was then closed in layers as usual. There were a few extrasystoles and periods of tachycardia when the pericardium was being opened and the ventricles sutured, but these proved transitory. The effect of the curare was neutralized by prostigmine 1.5 mg. intravenously and breathing commenced normally shortly after that. By 2 a.m. on 2 January the pulse rate had slowed to 112 per minute, with much improved volume and tension. A total of 2½ pints of blood had been administered throughout this period.

The next morning his pulse remained at 110 per minute, the general condition was much better, his respiratory rate had subsided to 34 per minute and by evening it was 28 per minute, with a pulse rate of 100 per minute. There was a slight increase in temperature during the next 2 days. This settled about the fourth day. From the time of admission, he was treated with penicillin and streptomycin. Physiotherapy consisting of respiratory exercises aimed at promoting full expansion of the lungs was started on the morning following the operation. On 3 January the blood pressure had risen to 110/75 mm. Hg.

Meanwhile an X-ray taken on 3 January 1958 showed no effusion in either pleural or pericardial cavities, and indicated a fully expanded lung (Fig 2). The water-seal drainage was discontinued on 4 January. An ECG was taken on 6 January. Both standard and chest leads appeared normal. This finding is quite compatible with a clean incised wound of the heart not involving the conducting system, unlike the findings in myocardial infarction. The patient was discharged on 15 January, exactly 2 weeks from the date of admission to the ward, and has not suffered any discomfort from his injury since.

Discussion

There are several points of interest in this case.

1. *Diagnosis.* The fact that there was a stab wound of the heart was not diagnosed before the operation. Looking back on the case, the only points that might have led us to suspect such an injury seem to be the weak pulse, the absence of a palpable apex beat, and the absence of any haemoptysis. Antero-posterior X-ray showed no evidence of fluid in the pericardial cavity. The weak pulse was, of course, due to cardiac embarrassment caused by the blood filling the pericardial cavity. Once the blood clot was removed the cardiac inflow improved considerably and the heart beat vigorously. The reason for urgent surgical intervention was the rising pulse rate and the respiratory embarrassment.

2. *Actual Anatomical Lesions.* The fact that blood was spurting from the wound of the heart at each beat, pointed to a penetrating wound of the ventricle. The lung and the inner surface of the chest wall were carefully inspected; there was no injury to the lung and no evidence of bleeding from the chest wall. This was further confirmed by the absence of fluid shown in the X-ray of the chest, taken a day after the operation.

3. *Route Traversed by the Weapon.* It entered the chest cavity between the fourth and fifth ribs in the midaxillary line, and was directed inwards, so that one would have expected the lung to be injured. The anterior surface of the left lung was presumably pushed back and so escaped injury. External measurements showed that the point of the weapon had traversed $3\frac{7}{8}$ inches (Fig. 3). The left cupola of the diaphragm, which is at the level of the fifth intercostal space on maximum expiration, would have escaped injury. The phrenic nerve, which passes over the left ventricle, was not damaged as it was much posterior to the level of the injury. The vertical level of the injury was that of the fourth left intercostal space at the sternal end, and from this it is possible to estimate the direction of the blow as being inwards, slightly forwards and somewhat downwards. As there was no bruising or abrasion at the external wound to suggest that the handle of the weapon was forced into the tissue, it is probable that the weapon used was a knife with a pointed blade at least 4 inches long, and a maximum width of 1 inch. If the victim was standing when the blow was delivered, the assailant, assuming him to be a right-handed man, would have been standing in front of him and slightly to his left. The injured man probably had his arm raised at the time he received the blow.

4. *Factors Involved in the Question of Survival after such an Injury.* If this man had died, one might have considered the injury necessarily fatal, and death to have occurred within a short time of the infliction of the wound. In this case the patient survived from about 3 p.m.

(when the injury was inflicted) until about 4.30 p.m. without any treatment at all. It is important to remember this type of case when reconstructing the sequence of events in death from injury to the heart; otherwise it may be difficult to explain the position of the body as found, when considered in relation to the available evidence about the place of assault. The factors which are usually responsible for death in injuries of the heart are free haemorrhage, interference with its action by the accumulation of blood in the pericardial cavity, or interference with the conducting system. In this case the rate of bleeding was comparatively slow, because the injury was in a muscular part of the heart, and was also probably oblique in nature. Cardiac tamponade was slight as there was free communication between the pleural and the pericardial cavities. Davidson and Fiddes[1] reported the case of a man who was stabbed in the heart but was able to walk a distance of 600 yards. He died about $1\frac{1}{2}$ hours after the injury. At post-mortem examination a penetrating wound half an inch long was found in the left ventricle. There was not much bleeding from the wound as it was oblique and produced a valvular effect. Since there was no communication with the pleural cavity, cardiac tamponade resulted. Lawrie[2] recorded a case of a penetrating wound in the left ventricle with haemopericardium and haemothorax. The patient recovered after operation. Ranasinghe[3] reported the case of a man who ran a distance of 200 yards after stabbing himself in the chest, and then collapsed. At post-mortem examination, cerebral embolism was found. The embolus probably originated in a thrombus found in the left ventricle attached at the site of a penetrating wound. There are several other such cases on record.

5. *Treatment.* There is much difference of opinion about the method of treatment in cases of this nature. There are two main lines of treatment, viz. thoracotomy (as advocated by Griswold and Drye[4] and Maynard *et al.*[5]) and conservative treatment with pericardial drainage where necessary. The latter method is recommended by Cooley *et al.*[6] and Chamberlain *et al.*[7] Cosman *et al.*[8] state that in a case treated by conservative methods, there was delayed bleeding resulting in haemothorax. In this case injury to the heart was not suspected as there was no haemopericardium demonstrable clinically or radiographically. They point out that it is necessary to have a definite period of observation when wounds of the heart are treated solely by pericardial aspiration.

6. *Prognosis.* Drye *et al.*[9] followed up a series of 20 cases of heart wounds over a period varying from 3 to 20 years. Of these, 12 were at work, and 6 had developed heart disease, which, however, could not be attributed to the injury. Lyons and Perkins[10] record a case of an aneurysm of the left ventricle with embolic episodes, 20 years after a stab wound of the heart. They discuss the literature on the subject and

express the opinion that aneurysms following stab wounds of the heart are rare. It should be pointed out that until quite lately recovery after such injuries has been rare. The results of recent cases can only be assessed after a sufficient period of time has elapsed since recovery from the injury.

Summary

A case of haemothorax following a stab wound of the heart, with recovery after operation, is described.

The diagnosis, the various lesions and the factors contributing to the survival of the patient are discussed.

Reference is made to recent articles discussing the treatment of this type of case, and its long-term prospects.

Our thanks are due to Mr. P. R. Anthonis, Surgeon, General Hospital, Colombo, for permission to submit this case for publication. Thanks are also due to Dr. C. M. H. Gunaratane, Anaesthetist, but for whose skill and care this operation could not have been successfully carried out, and to Drs. G. W. Karunaratne and R. Amerasingham, the Senior and Junior House Surgeons, for the prompt and effective pre-operative measures adopted.

References

1. Davidson, J. and Fiddes, F. S. (1956): Brit. Med. J., **1,** 210.
2. Lawrie, R. W. (1955): Brit. Med. J., **1,** 952.
3. Ranasinghe, H. (1957): J. Forensic Med., **4,** 128.
4. Griswald, R. A. and Drye, J. C. (1954): Ann. Surg., **139,** 783.
5. Maynard, Aubre De L., Avecilla, Marcelline J. and Naclerio, Emil A. (1956): Ann. Surg., **144,** 1018.
6. Cooley, D. A., Dunn, J. R., Le Roy Buckman, H. and De Bakey, M. E. (1955): Surg., **37,** 882.
7. Chamberlain, J. M., Canberry, D. M. and Stefko, P. L. (1956): Amer. J. Surg., **91,** 600.
8. Cosman, B., Byerly, W. G. and Wickman, W. A. (1958): Ann. Surg., **147,** 87.
9. Drye, J. C., Coe, W. S. and Stamer, John P. (1956): Amer. J. Surg., **91,** 597.
10. Lyons, C. and Perkins, R. (1958): Ann. Surg., **147,** 256.

Review of Books

THE DOCTOR-PATIENT RELATIONSHIP

Privileged Communications between Physician and Patient. By Clinton DeWitt. 1958. (Pp. 502 + Index. 87s. 6d.). Oxford: Blackwell Scientific Publications.

This is an erudite, comparative, historical study of the numerous ways in which the professional relationship between a medical practitioner and his patient has developed special duties and privileges.

The confidential relationship is not unique to physicians. It obtains also between a lawyer and his client as well as a priest and the members of his flock. The physician, however, is more likely to find his professional obligations tested and tried because he may become involved in issues raised before the courts. The ethical duty to observe secrecy is enshrined in a long and honourable tradition crystallized as a principle at least as far back as 400 B.C., when Hippocrates vowed, *inter alia*:

'Whatsoever things I see or hear concerning the life of men, in my attendance on the sick or even apart therefrom which ought not to be noised abroad, I will keep silence thereon, counting such things to be as sacred secrets.'

Medical practitioners and their patients, however, do not enjoy that immunity which has been accorded lawyers in their professional relationships with their clients. But the ethical pressure not to breach the patient's confidence is so great that no medical practitioner is likely to disregard it unless by express direction of a Court. If he were to disregard his professional obligations or to treat them with irresponsibility, he would expose himself, apart from any other risks, to disciplinary action by the statutory bodies governing the ethical standards of medical conduct. In England, e.g. this body is the General Medical Council or the General Dental Council; in South Africa it is the South African Medical and Dental Council.

The situation in the United States is primarily the scope of the present work, the like of which has not heretofore been attempted. The author's reasons for and purpose in writing this work are admirably set out in the *Preface* at p. vii.

'This treatise is the outgrowth of 30 years of experience in trial and appellate courts, both federal and state. It is my opinion that members of the Bench and Bar have too often misunderstood the paramount purpose of the (physician-patient) privilege, with the lamentable result that persons not within its intended scope have been permitted to invoke its protection, and that material and relevant evidence concerning communications and information not within the ban of the statute has been excluded. Furthermore, the doctrine of waiver has often been improperly applied, or overlooked entirely. I have endeavoured, therefore, to produce a comprehensive yet practical treatise which will be helpful to the trial lawyer and the trial judge in solving many of the problems that constantly arise from this troublesome and much abused rule of evidence. I hope, too, that medical men and teachers of law will find it beneficial. No textbook dealing exclusively with this testimonial privilege has hitherto been available. Due to the dissimilarity of the statutes which create the privilege and to the disparate attitudes of the Courts toward it in general, the great mass of reported cases presents a formidable task to any author who would attempt to write a treatise on this subject. This, however, I have undertaken to do.

An appendix is included wherein appear the existing statutes pertaining to the privilege; and also the appropriate rules which form a part of the Uniform Rules of Evidence. Reference to these, I believe, will readily enable the reader to acquire a better understanding of some of the cases which are discussed in the text and in the notes of this treatise.'

The intricate situation which can arise in the U.S.A. in connexion with autopsy findings is admirably set out on pp. 130–134. The position, of course, varies in different countries. In South Africa, e.g. statutory provision is now made for all autopsies, whether or not death has been due to natural causes. Although a dead body is a *res nullius*, the ethical rule of the Medical Council nevertheless enjoins the following obligation:

'*Professional Secrecy*: Divulging verbally or in writing any information which ought not to be divulged regard-

ing the ailments of a patient except with the express consent of the patient or, in the case of a minor, with the consent of his guardian, or in the case of a deceased patient, with the consent of his next-of-kin or the executor of his estate.

Note. In a court of law, professional secrecy should be contravened only under protest after direction from the presiding judicial officer.'

It is certainly not clear what meaning attaches to such an undefined phrase as 'next-of-kin.' The rule, however, reflects the concern of the statutory disciplinary body to maintain professional secrecy after the death of a deceased person, even if he was not, during his lifetime, a patient of the practitioner performing the autopsy.

Litigation involving the practice of medicine has reached a peak never before attained in the history of society. The role and the functions of medical practitioners have accordingly also attained an importance which was probably unsuspected even 20 years ago. Professor De Witts' monumental treatise covers the problems that may arise in civil and criminal cases. His exhaustive study should provide a guide to almost every conceivable contingency. It is a 'must' for the lawyer. For medical practitioners it also certainly falls in the category of required reading.

Courtroom Medicine

Courtroom Medicine. Compiled and Edited by Marshall Houts. (Pp. 505 + Index. Profusely Illustrated. £5 5s.). Oxford: Blackwell Scientific Publications.

Courtroom Medicine has been written by a distinguished band of experts who include anaesthetists, surgeons, ophthalmologists, physicians, radiologists, anatomists, psychiatrists, obstetricians and gynaecologists, as well as legal practitioners.

The volume deals with almost every conceivable practical aspect of the ways in which medical practitioners may today become involved with the law in the course of their professional duties. The first three chapters, which outline the nature and functions of the medical expert witness, deserve the most careful study not ony by doctors but also by medical students.

The chapter on *Some Fundamentals of Anatomy* will be of great interest and assistance to lawyers, particularly because of the instructive illustrations which will materially assist them in understanding their problems during preparation and consultations and presenting their material during a trial. The chapters on *Allergy* and *Anesthesia* are very timely because of the increasing litigation with which hospitals and medical practitioners are faced to-day, in respect of actions connected with these fields of practice.

The evaluation of physical disability is ably reviewed in the chapters on *Back Injuries, Bones, Joints and Ligaments, Disability Evaluation* and *Joint Disability.*

It will surprise most readers to find that a chapter of almost 30 pages is devoted to the *Medicolegal Aspects of Cancer* (exclusive of the discussion on the relationship between trauma and malignant disease elsewhere in the book). The importance of occupational cancers is fully reviewed and the role of trauma in activating cancer is thoroughly canvassed. This whole field of inquiry has possibly been unduly neglected in the past and it will repay careful study. It certainly opens up a profitable area for investigation and research.

Head Injuries occupy a very important part of the book. There is a very good critical survey of the incidence of post-traumatic epilepsy in relation to open and closed head injuries. A. Earl Walker expresses the view that if epilepsy has not developed within 2 years after a closed head injury, the chances of subsequent fits are probably as low as 1%, which is just slightly more than the incidence of epilepsy in the general population (p. 196). In the case of open head injuries, there is about an 8% chance (from the end of the second year to the tenth year) of the patient's developing attacks.

About 2–5% of head injuries can be expected to develop attacks after 10 years from the time of injury.

The prognosis is also dealt with very clearly. Warren's own findings indicate that there is almost a 40% chance that a patient having a few attacks in the first 2 years may be kept free of attacks by adequate medication (p. 197). He found that after the 2nd year 27% had no further fits of any type and 13% rare or insignificant attacks.

Radiation Hazards are reviewed by Marshall Brucer, M.D., Chairman of the Medical Division of the Oak Ridge Institute of Nuclear Studies.

There are also chapters on *Toxicology* and the newer investigative procedures such as *Aortography* and those involving the *Cardiovascular System.*

Dr. J. E. Schmidt has prepared a most valuable dictionary of explanation of medical terms (*Some Medical Terms Defined for the Layman*). This occupies

over 130 double-column pages and is itself an important reason why this volume should be on the bookshelf of every legal practitioner.

No book can replace the consultations between the lawyer and the medical expert. The present volume, however, will facilitate the discussions, guide the legal practitioner to the crux of the issues and assist the medical witness in understanding what the courts require of him.

Courtroom Medicine is a unique contribution to our forensic literature. Its truly encyclopaedic and authoritative character makes it an essential volume for all who have to deal with the numerous medical problems which come before our courts.

Abstract

Encephalopathy Following Minor Injury

Serious cerebral lesions may be detected by thorough histologic examination and may be much more significant than meningeal haemorrhage or small contusions prominent on gross examination.

John Denst, M.D., and associates of the University of Colorado, Denver, describe a previously healthy 17-year-old boy who struck his head in a wrestling match, lay motionless and mute for 2 months, became fairly alert but unable to co-operate, and died 13½ months after injury.

Autopsy showed extreme demyelination throughout the white matter of the cerebral hemispheres and extensive cortical atrophy.

[Arch. Path., **67**, 134, 1959].